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Original Articles

EXPERIMENTAL STUDIES OF RHYTHM, ANXIETY AND CEREBRAL VIGILANCE

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In a recent book, Sargant (1957) has discussed many examples of human beings who, after brief or prolonged mental tension, including terror, underwent a sort of reactive collapse into a state which, in its extreme form, was one of stupor. Sargant interpreted these reactions in terms of a physiology derived from Pavlov, particular emphasis being laid on "transmarginal inhibition"—a variety of the "internal inhibition", or "hypnosis" which Pavlov identified with sleep, and which was manifested by motor inertia and unresponsiveness to stimuli. Pavlov's dogs exhibited internal inhibition in response to monotonous stimulation of any kind (including repeated electric shocks); repeated non-reinforcement, disappointment, or non-fulfilment of the dog's expectations; delay in reinforcement; and, in experienced dogs, in response to being placed in the experimental situation (or having the stimulating electrode attached to the leg in the case of those given electric shocks).

Pavlov's theories bear little recognizable relation to contemporary neurophysiological knowledge. It would appear desirable that his observations, and the phenomena discussed by Sargant, should be related to our present knowledge of the mechanisms governing sleep and wakefulness, if Pavlov's views on the identity of internal inhibition and sleep are accepted. It should, meanwhile, be borne in mind that Pavlov concluded that internal inhibition, or sleep, could be localized to certain groups of functions, or certain areas of cortex.

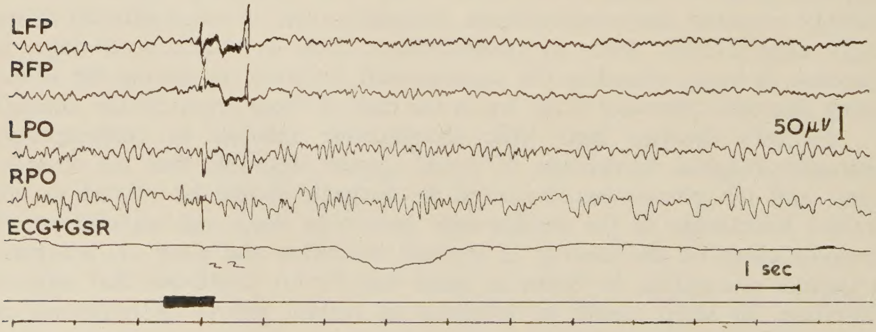
Some laboratory experiments with human subjects, from whom electroencephalographic and other variables were recorded, and in whom signs of internal inhibition appeared, will be outlined below and discussed in the light of the previously mentioned considerations.

I. An Initial Experiment

In the course of an exploratory experiment, carried out for purposes discussed elsewhere (Oswald, 1958), a healthy 19-year-old male volunteer of exceptional courage was exposed, in 13 experimental sessions, spread over several weeks, to three different, brief auditory tones, given in irregular order, at irregular intervals of 2-40 seconds. One of the tones, on about half of all occasions, was quickly followed by a very bright flash of light and a momentary electric shock to the leg, of an intensity sufficient to cause considerable emotional arousal.

In the first six experimental sessions, there were 496 presentations of the "significant" tone and 253 of these were followed by the shock and light. These sessions were spread over nine days, during which the subject became increasingly apprehensive and his sleep at night was disturbed by nightmares involving shocks, lights and the experimenter. During the sixth session, hallucinations of light and shock occurred for the first time, as conditioned responses to the "significant" tone. The subject was, however, aware in this session that these hallucinatory experiences were not real (in later sessions he was unable to differentiate between the real and hallucinatory stimuli). He became distressed and the experimental session was interrupted and, soon after, prematurely ended because of his distress.

The subject then went away on sixteen days' holiday. On his return, having had time to contemplate the renewal of his ordeal, he was extremely apprehensive, but was persuaded to submit to a further session. He lay on a couch in a normally lit room, exposed to many intermittent noises both from inside and outside the room. In this session, the light was unchanged but the shock was reduced in strength, though it still caused flinching, tachycardia and a galvanic skin response. Within five minutes of the start of this session, the EEG revealed the pattern of sleep with generalized activity at 5–6 c/sec., bursts of 14 c/sec. and some 20 c/sec. waves. This continued for six minutes, after which the session was ended. In this period there were 32 tones, of which fifteen were "significant", nine of the latter being followed by shock and light. Fig. 1 shows how briefly the electroencephalographic sleep pattern was disrupted by the powerful shock and intense light. When the session ended, the subject had to be awakened,



LFP=left fronto-parietal RPO=right parieto-occipital
RFP=right fronto-parietal ECG+GSR=electrocardiogram with galvanic skin response.
LPO=left parieto-occipital

Fig. 1.—Tone at marker, and shock and light at artifact. The stimuli cause return of alpha rhythm, a galvanic skin response and tachycardia, but sleep patterns then recur within 5 to 6 seconds. A pulse artifact is present in RPO.

whereupon he declared he had been asleep and having dreams which involved music. He had only vague memories of receiving shocks.

In this session shocks had been administered by the discharge of a 0·25 microfarad condenser, charged to 290 volts, through the subject's leg, using EEG electrodes, the resistance between which was approximately 50 kilohms. In this session, though not in previous sessions, each side of the condenser, when shocks were given, was also connected through 15 kilohm resistances to earth.

Comment

It was thought, after this unexpected finding was encountered, that the sleep had appeared because the stimulus of principal significance, though still powerful, had not been sufficiently strong to fulfil the subject's worst fears, and that his very great emotional tension had been succeeded by a relief so great as to amount to sleep.

In his case, stimuli had been given irregularly, so that he was never given periods of freedom from anxiety from one stimulus to the next. It was postulated that if tension, or anxiety, were to be produced by some stimulus which recurred regularly, then tension would be reduced between presentations of that stimulus, and sleep might appear at such times.

II. EXPERIMENTS WITH REGULARLY RECURRING STIMULI

These experiments fell into two main groups, the results in each group being substantially the same. In one group, anxiety, or tension, was provoked by the regular presentation of electric shocks. In the other group, the subjects accepted an obligation to attend, to the limits of individual ability, to very faint, regular, sensory stimuli.

(a) REGULAR ELECTRIC SHOCKS

Subjects and Method

Six normal, healthy volunteers, aged 19-30, none of whom had volunteered with knowledge of what might be asked of them, and none of whom knew the purpose of these experiments, were persuaded to submit to electric shocks. They were not sleep-deprived.

The experiments were carried out with the subject lying on a couch, with eyes closed, in two cases in a normally lighted room, and in the other cases in a dimly lighted room. A cap held EEG electrodes in place. Usually, the respiration, using a mercury-in-rubber tube round the chest, and also the electrocardiogram and the electrodermogram, using EEG electrodes on the palm of a hand and one leg, were recorded. The subject wore headphones, and, at regular intervals, which varied between 9-17 seconds in different sessions, was presented with a clearly audible tone of about 800 c/sec. This tone was of fixed duration in any one session, about two seconds. When the tone ceased, the electric shock was given by discharge of a 0·1 microfarad condenser charged to 320 volts. This was discharged through two EEG electrodes strapped to the wrist or ankle. At the end of each session, in which up to 180 shocks were given, the resistance between the electrodes was measured. The electrodes were always applied with electrode jelly to skin which had been de-greased with alcohol and lightly scraped with a sharp edge. The amount of noise to which the individual was exposed varied considerably from session to session. One subject had 4 sessions, three had 3 sessions, one had 2 and one would only submit to one session.

The first few shocks of each session were reduced by means of a potentiometer in series, but this resistance was reduced to zero after the first dozen or so shocks. A few kilohms in series make the shock feel much weaker than does an equal increase in skin resistance.

Results

Four of the subjects showed EEG signs of sleep between the shocks. In one case sleep deepened, being broken only by K-complexes with each shock (fig. 2). In another subject, sleep signs appeared in the EEG during the intervals between shocks for the last 100 of the 173 shocks of his 3rd session. In another, during the intervals between the 30th to the 60th shock, of 73 given in his second session. In another, during the intervals between the last 110 shocks of the 170 given in his third session. The subject who had only one session did not show EEG sleep signs.

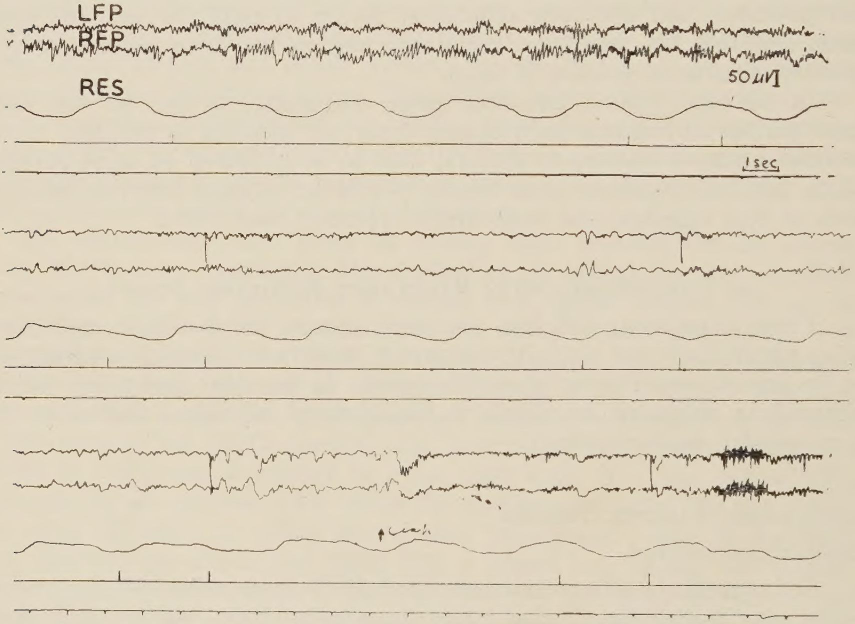


FIG. 2.—Subject X.

RES=respiration (inspiration down) with apex beat movements visible. Other abbreviations as in fig. 1. The first of each pair of marker signals indicates the start of the warning tone. The second indicates the end of the warning tone and the moment of the shock. The resistance between the electrodes at the end of the session was 20 kilohms. The top excerpt shows the 4th and 5th shocks (heart rate 75/min.), the middle excerpt shows the 123rd and 124th shocks (heart rate 56 to 75/min.), the bottom excerpt the 164th and 165th shocks (heart rate 56 to 80/min.).

The transition, from alertness to light sleep, with brief arousal at each stimulus, to deeper sleep with only K-complexes in response to stimuli, is shown. The clap causes a K-complex and 12 c/sec. waves, the next shock causing full arousal. The change in the form of the respiratory curve when sleep appears and when sleep ends is clearly shown.

The remaining subject was one who had seemed particularly apprehensive. In his case, though he did not show signs of sleep between shocks, yet in his third session, after 40 shocks, the shock-producing apparatus failed and he was told he would get no more shocks for a while, whereupon, within half a minute, he went to sleep. He woke up when the shocks restarted, but again went to sleep when another apparatus failure occurred later.

Apart from dreams, subjects described feelings of derealization. This was true, for instance, of the man just described. In common with the other subjects, his galvanic skin response (GSR) showed habituation to zero after a sufficient series of regular shocks. In his first session, after 45 shocks, it was noticed that the strength of the shock was not maximal. It had, inadvertently, not been increased from the modified strength always used for the first dozen shocks of each session. The shock-strength was thereupon increased to maximum. The GSR which had disappeared, did not, however, reappear. Nevertheless, after the session was over, the subject commented on the increase of shock-strength which had occurred about half-way through and said he had been quite aware of it, but only as if he were a "detached observer".

In all the subjects, when the GSR had shown habituation to zero, any novel sensory stimulus, such as an unexpected noise, would be followed by the reappearance of a GSR for several shocks.

Hypnotic analgesia

Two subjects, one of whom had been used in the experiments just described, and a further healthy, 19-year-old male, both of whom could attain fairly deep hypnotic trances, received extremely strong electric shocks to the arm following suggestions of analgesia during hypnosis. The same apparatus was used as formerly, but by dint of abrasion sufficient to cause a little oozing of serum, the skin resistance was reduced to only 500 ohms approximately.

The subject with the previous experience of receiving shocks, now received, during suggested analgesia, 390 in one session, at 12-second intervals without any warning signal. Despite denial by the subject of discomfort, large GSRs occurred at first, but gradually declined. Occasionally, absence of the GSR, and also EEG sleep signs between shocks were noted. This was especially so if suggestions of relaxation, sleep, and no pain, had just been made.

The second subject with hypnotic analgesia had no prior knowledge whatsoever that he was to receive electric shocks. He had previously volunteered to take part in any hypnotic experimental work. He had very large GSRs to the first shocks, but, despite the fact that, in his case, no further suggestions were made to him, the GSR showed habituation to zero after 60 shocks. In his case therefore the reduction of the GSR may be attributed to habituation rather than hypnotic suggestion.

(b) Regular Intent Listening

In these experiments twelve healthy male subjects aged 19-24 were used. In addition, a male patient, aged 51, who had a history of cataplectic attacks on laughter or anger for over 20 years, and of attacks of sleep, especially when bored—in buses, lectures, etc.—but also at other times, even when out walking, for three years.

The subject, in each case, lay, with eyes closed, on a couch in a normally lighted room, with a switch for signalling responses in one hand. A head cap held EEG electrodes in place. There were three electrodes on each side of the scalp, about 5 cm. from the midline, in posterior frontal, parietal and occipital positions, so that rhythms from rolandic, as well as more posterior areas could be recorded. Usually the respiration, and the electrocardiogram were also recorded.

At regular intervals, auditory signals, consisting of pure tones of approximately 700 c/s, were presented, through a loudspeaker in a few early sessions, and through padded headphones in later sessions. The subject was required to click his switch if he heard the tone.

The intensity of the tone was controlled by a dial of an oscillator. The intensity of the tones was gradually reduced in the first five or ten minutes, till a level was found at which about half were missed. The intensity was then slightly increased, though, as will be described, signals would once more be missed as the session wore on. In all sessions occasional tones were deliberately withheld. The tones were given at intervals of 10, 5, or 3 seconds during sessions lasting, generally, 15-30 minutes.

The EEG Findings

The most striking finding, in these situations where the subjects' task was to exercise a high degree of alertness, was the insistent tendency for the EEG to take on the appearances of sleep.

In the typical session, at the time of each of the first few tones, the alpha rhythm would block for a couple of seconds, and then return throughout the waiting period; soon, however, the alpha rhythm would be present continuously; later a "reversal" of the initial picture would occur, namely a flat record with or without low voltage slow waves between signals, and a burst of alpha rhythm at the time the signal was about to occur, or especially if sleep signs had included slow waves, during and after the time of response. This burst of alpha rhythm might then die away, the record flattening again, or else the alpha rhythm would be present continuously for a period, till, once more, it would die away. Then, for several signals, it might appear only at the time of the signal, as in fig. 3. Then it might not appear at all for a couple of signals, and to these no response would usually be made. Then, either apparently spontaneously, or in response to some extraneous noise (see fig. 9), or at the time the next signal was due, the alpha rhythm would suddenly return. Sometimes, as will be discussed in detail later, sleep signs would continue during responses, except in one or both rolandic areas.

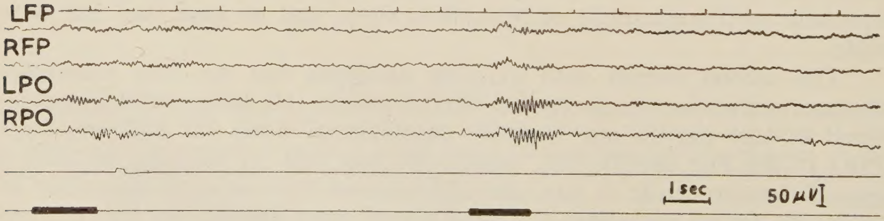


FIG. 3.—Subject A. The bottom marker indicates the faint tone and the subject's response is on the channel above.
Illustrating the typical and constantly recurring picture of EEG signs of light sleep between stimuli. Notice that arousal occurs at the time of the second stimulus shown, though it was not heard (i.e. not overtly responded to).

There seemed to be two tendencies at work, one, a tendency for sleep and alertness to alternate regularly at the rate of the signals, the other, a slower general "downward" drift of variable steepness and duration towards sleep. The latter drift might go so deep as to be associated with failure of response. The ending of the downward drift was usually abrupt, and alerting would be followed by another such drift. Subjects would occasionally remark, sometimes after a slight start, "Sorry, but I was asleep", and would later report dreams. As a rule, however, subjects had no realization that they had missed many of the signals. These failures to respond to the signals tended to occur towards the end of experimental sessions, when the regular alternation between sleep and alertness at the rate of stimulation had become rather lost in slower sleep drifts (see fig. 4).

It is hardly practicable to quantify the appearance of EEG sleep signs in the records of all subjects, but Table I may give an indication of the differences between individuals. There were differences from session to session in the same individual, as well as differences between individuals. In the table, the term "gross" indicates that the subject showed the type of record seen in fig. 4 for periods of several minutes in a session. The term "marked" indicates that the subject showed the sort of changes seen in fig. 3 for several minutes in a session. The term "mild" indicates that those changes occurred between only a few signals in the session. Subject E was the patient with narcolepsy. His records showed no great difference from those of the other subjects who showed "gross" sleep signs, except that in his case, such signs appeared very quickly in each session.

TABLE I

Subject	Total number of sessions	Sleep signs	Sessions in which sleep signs first seen
A	17	gross	3rd
B	3	gross	1st
C	3	gross	1st
D	2	gross	1st
E	2	gross	1st
F	2	gross	1st
G	2	marked	1st
H	2	marked	2nd
K	1	marked	1st
L	3	mild	3rd
M	2	nil	—
N	1	nil	—
P	1	nil	—

In order to illustrate the course of events, a typical session may be described—the first with subject C.

The subject, a 22-year-old male, healthy, athletic and university educated, lay on the couch in the EEG room, the outside of which was being painted. Throughout the whole session there was a constant succession of talking, shouting, whistling and singing from the painters.

The tones were delivered without a warning at 10-second intervals through headphones. The tones were made progressively fainter, then slightly louder when the threshold seemed near, and after the first 20 tones the intensity was held constant. After the 26th tone his usually fairly continuous 30-40 microvolt alpha rhythm began to disappear between tones, and this flattening became more and more obvious, with many slow waves, till the 50th tone was missed, having occurred during some 4 c/s waves of 30 microvolts. Arousal then occurred, and sleep signs did not reappear for another 8 tones, two of which were missed when they coincided with loud outside noises. Sleep signs then returned between tones, till after another 33 tones, the next tone was missed during a sleep drift. Arousal occurred again but 10 tones later another was missed during sleep. Then one was responded to, then 3 missed in a row. Then he got 3, then missed another 3, then got 6, missed one, got one, missed one, got one, missed one, got one, missed one, got one and then the sequence shown in fig. 4. Twenty seconds later he gave a violent bodily jerk of the type which may occur in light sleep (Oswald, 1959c).

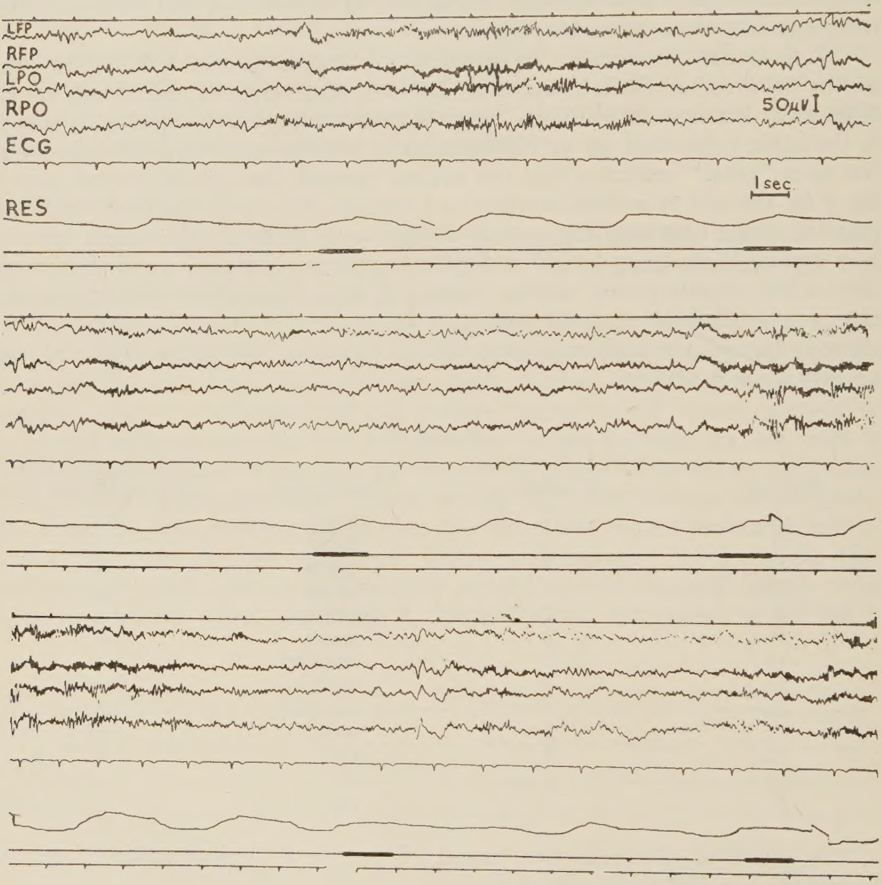


FIG. 4.—Subject C. The excerpt is continuous from above down. The response marker is superimposed on the respiration channel, the stimulus marker being on the channel below.
Showing longer sleep drifts and how the stimulus is "above threshold" when 10 to 12 c/sec. waves are present but not when sleep rhythms are present.

Afterwards he said that he had "lost concentration at times", though he had not been asleep "except just when I jerked—I'd been dreaming then about being in Edinburgh with my girl friend and about a Rugger match". After the jerk "I came out all over in a sweat, suddenly realized I must have been asleep and started listening like hell for the buzzer". Of the painters, he said he got very annoyed with them, wishing they would go away so that he could hear the faint tone more easily. He had indulged in fantasies in which he harangued them, telling them to be quiet, though "later it didn't bother me". He had found himself "thinking" and "having visions", and "then I would keep getting back to the job".

In three of his last four sessions, the highly experienced subject A, after sitting on a chair while the head electrodes were attached, went immediately and obviously (behaviourally at least) to sleep when he lay on the couch, waiting for wires to be attached and the experiment to begin (shades of Pavlov's dogs!).

COMMENTS ON (a) AND (b) ABOVE

The "threshold"

If fig. 4 is inspected it may be seen that whether or not the subject responded, whether or not the faint stimulus was "above threshold", whether or not what some would call a "voluntary" conditioned reflex of switch-pressing occurred, depended on the degree of wakefulness of the subject. The subject of fig. 2 expressed annoyance about the clap because, after it, the shocks were again felt as severe. It may be recalled that the frequent failure of response, in subjects required to attend continuously for minimal stimuli, led Mackworth (1950) to the conclusion that internal inhibition was responsible.

The regular recurrence of sleep

In EEG studies, Loomis *et al.* (1937) and Gastaut *et al.* (1957) have reported the regular alternation of sleep and wakefulness when stimuli have required responses at one-minute or half-minute intervals. In the present experiments, both with electric shocks at 9–17 second intervals, and with faint auditory stimuli at 10-second intervals, similar pictures of alternation between sleep and wakefulness at these rates have been seen. The same was true, moreover, if stimuli were presented every 5 seconds or every 3 seconds (figs. 8 and 5).

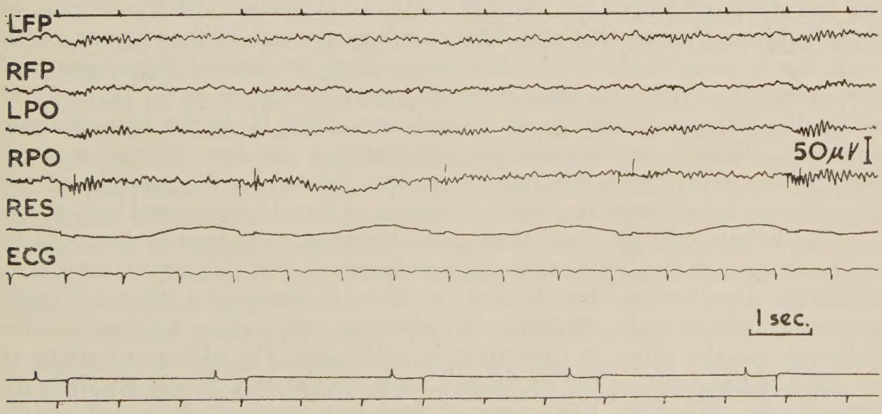


FIG. 5.—Subject C. The response marker is again superimposed on the respiration channel, the stimulus marker being at the bottom.
Showing alternation of alpha rhythm, and a flat record with low voltage theta waves, occurring regularly at intervals of just under 3 seconds. Note also the synchronization of respiration rhythm with the response rhythm.

The galvanic skin response

In the experiments with regular electric shocks, habituation of the GSR was noted. The skin potential response (Tarchanoff response), not the skin conductance change (Féré response) was recorded. Seward and Seward (1934), and Hardy *et al.* (1952) reported decrease of the GSR with repetition of electric shocks, but not actual disappearance, though the GSR of one of the subjects of Hardy *et al.* became very small.

Neither of these groups of workers used nearly so large a number of shocks in any one session as was used in the present experiments. They were, it should be added, recording the Féré effect, but the evidence is that the Tarchanoff and Féré effects are always parallel, in the sense that if a stimulus is effective in producing one, it will produce the other (Duret-Cosyns and Duret, 1956; Montagu, 1958).

The respiration

Two modifications of respiration were seen (a) of the form of the respiratory curve, (b) of the rhythm of respiration.

(a) Magnussen (1944) studied the respiration during sleep. His subjects had to wear a face piece and breathe out against the resistance of an expiratory valve. Under these special conditions, he found an increase of the expiratory phase during sleep, and mentioned that, by this means, very brief episodes of sleep could be detected. He believed that, during sleep, expiration became purely passsive, and hence, owing to the resistance of the valve, became prolonged. Under recording conditions he used, the movements of the chest did not show characteristic changes during sleep.

The mercury-in-rubber device used in the present experiments gave a distorted respiratory curve, owing to the short time-constants of the EEG machine (a 0·3- or a 1·0-second time-constant). The effect was to mask slow chest movements and hence emphasize rapid chest movements. Towards the end of the experiments it was noticed that, in fact, the recording method did show a quite characteristic difference of the respiratory curve, between alertness and sleep, as judged by the EEG signs. Examination of the records of all the subjects showed that this difference was present in the records of all except one of those subjects who had shown gross or marked sleep signs, and whose respiration had also been recorded, namely subjects A, C, D, E, G, H and X. It was not apparent in the records of subject B. In the case of those subjects in whom it was present, the phenomenon was less obvious in some sessions than others, and this may have been related to the position of the tube, whether it was recording largely thoracic, or largely abdominal movement.

The difference in the respiratory curve between alertness and sleep can be very easily seen in fig. 2. The most obvious feature is that the gentle respiratory undulation alters during sleep, so that expiration is shown as a sharp and rapid movement, which ends abruptly. In addition, respiration becomes rather shallower and the pause in expiration is prolonged. The change of shape of the curve at the onset or end of sleep is quite abrupt and closely parallels the EEG changes. When the EEG signs of sleep and alertness alternated rapidly every 5 or 3 seconds, the changed appearance of respiration was not very apparent.

This finding was unexpected and incidental. It suggests that if expiration during sleep is passive, then during the waking state, not only is the musculature used to enhance the force, if necessary, but that it also smooths expiration. Apparently, Aristotle stated that sleep was characterized by very abrupt expiration (according to Kleitman, 1939), but most workers seem to have been concerned with regularity and depth of respiration rather than its form.

(b) The respiration showed a tendency, variable between different subjects and between different sessions with any given subject, to synchronize with the rhythm of stimulation. This synchronization never persisted during long sleep drifts. The synchronization would persist from one to a dozen or more stimuli, sometimes also apparently occurring as a two breaths to one signal rhythm. The longest single respiratory cycle seen with synchronization was 18 seconds, and the shortest, as in fig. 5, of 3 seconds (but see fig. 7).

The effect on the rhythm of respiration was by no means unexpected, and has been described by others in connexion with studies in which regular auditory stimuli were given (e.g. Lovell and Morgan, 1942).

The heart rate

Cyclical changes of heart rate, at the rate of stimulation occurred. Generally the heart quickened just after the response switch was pressed, or the shock received, and then slowed during the waiting period, especially if sleep appeared. Occasionally, however, during the listening experiments, the cycle was one in which the heart slowed suddenly at the expected moment of the faint stimulus.

III. MOVEMENT TO JAZZ RHYTHMS

In the course of the experiments which have already been outlined, subjects who were exposed to, and responded to, rhythmic stimuli, manifested states of altered awareness, altered emotional responsiveness and (as will be discussed subsequently) states of "partial" sleep. Sargant (1957) paid special attention, in his book, to the role of prolonged movement and dancing to rhythmic stimuli, in facilitating various trance states of altered awareness and responsiveness, with suggestibility.

It was therefore a logical extension of the previous experiments, to attempt an EEG investigation of persons responding to more rapid rhythmic stimuli, which took the form of jazz music, in order to maintain co-operation and interest. Subjects get fed-up with dull, difficult, monotonous tasks, just as Pavlov's dogs exhibited "negativism".

It was of particular interest to note (1) whether EEG signs of alertness and sleep would alternate at the relatively rapid rate of the rhythm, (2) if a general tendency for the appearance of prolonged sleep signs would occur, (3) if respiration would tend to synchronize with the rhythm. Ellis and Brighouse (1952) found a significant rise in respiration rate when people, engaged on a reading task, were exposed to five-minute records of Liszt's "Hungarian Rhapsody No. 2" or a blues record, but a soothing classical selection was less effective.

It was not possible to perform EEGs on people actually dancing. However, it was possible to get subjects to sit, or lie, and move in time to the music, which, in some cases, provoked real pleasure, even approaching that state, apparently attainable without recourse to alcohol, known to jazz-lovers as being "sent".

The experimental method and the subjects

Three tape recordings, two of traditional jazz, known as "blues", and one of "hot swing", were prepared. The first (Tape I) was specially recorded by an Oxford undergraduate band. It consisted of continuous blues music with loud drum bangs every twenty seconds. Tape II was prepared from gramophone records made by two of the world's leading jazz bands. The assortment of "blues" was made up of items lasting 5-10 minutes each, each of which had a different rhythm, and between which there were momentary discontinuities. Tape III was recorded from a long gramophone record of an "All Stars Jam Session"—a fearsome effort. Each tape recording lasted approximately one hour, and was always played loudly.

The subjects available were some of those who had been used in the experiments of Section II above. In some sessions (8 in all) the subject would lie on a couch, moving on one or both sides of the body to the music—this involved moving (say) the right foot in time with the music, and, with the hand of the same side, pressing a switch in time with the music. The switch was deliberately a large and awkward thing to hold in one hand, and required a thin, firmly sprung lever to be pushed forward, and released, in time with the music. The record of the switch operation therefore was a record of performance of a more skilled nature than that involved in the leg movement. On other occasions (12 in all) the subject would sit up in a chair tapping both feet and banging both hands vigorously up and down.

Apart from four EEG channels, the electrocardiogram, the respiration, and the movements of at least two limbs, either via the switch, or by placing a pair of electrodes over muscles of the limb, were recorded.

Two of the eight subjects were jazz fans (one made a practice of attending all-night "jam sessions" and other jazz concerts). Four others liked jazz and appreciated the music used in the experiments. One other liked jazz but was irritated by the technical deficiencies of the musicians in his only session (Tape I). The remaining subject, the narcoleptic patient, though sufficiently co-operative to take part in two sessions, did not like jazz.

Results and Comments

Sleep Signs

The EEG changes during continuous movement to the jazz were essentially similar to those described in the experiments of Section II. Signs of light sleep were seen for periods of up to a minute or more, e.g. fig. 6, in total, occupying 20–50 per cent of the total recording time of the sessions in the case of six of the subjects. The total duration was scarcely possible of exact determination, because, apart from the subjective nature of the decision where the signs were of borderline drowsiness, so many of the sleep episodes were exceedingly brief. One subject showed only occasional signs of drowsiness, lasting for periods of about 10 seconds or so. The subject who was irritated by the technical imperfections of the musicians, showed no long episodes suggestive of drowsiness, but his alpha rhythm appeared to wax and wane in time with his movements. Three other subjects showed a similar tendency even more clearly (this can be seen in fig. 6).

"Relief" and sleep

It was striking how, after a period of raucous cacophony by the band as a whole, when perhaps the rhythm was a little difficult to follow, sleep signs would appear if there now followed a clear and pleasing clarinet solo with a background of regular drum beats (fig. 10). This was especially marked with Tape II, in which there were many solos by a world-famous clarinettist. These clarinet solos were often experienced as quite delightful. These, and one other particular passage, produced the nearest approach to ecstasy. This one other passage was the only piece in which anything other than music was heard, for in it there were bursts of applause, and the band leader's familiar croaking voice shouting comments. It was remarkable that during these episodes of cheering and shouting, the EEG records of three of the subjects, including the two jazz enthusiasts, showed particularly striking sleep signs, so much so that on two occasions I stopped recording and questioned them about their experiences. As Subject C put it, "It gives you a feeling."

We may thus say that sweet music following loud and raucous passages, and the sense of being with others in appreciating the music, both induced feelings of delight, with inner peace, and EEG signs of sleep—as if the individuals were experiencing dreams, the material of which was largely provided by an external source. Ecstasy necessarily must involve withdrawal from prosaic reality; the sense of being with others no doubt helped subjects to escape from the mundane laboratory surroundings of reality to a dream-world.

It should not be thought, however, that the appearance of sleep signs depended wholly on relief provided by sweeter music, or a feeling of being part of a crowd. The two jazz enthusiasts showed long episodes of sleep signs during the loudest and most violent passages of Tape III, which was of what might be described as uninterrupted virulence—they were the only subjects willing to listen to this.

It was of interest that those subjects who moved to the music, both while sitting up in a chair, and lying down, showed just as great a tendency to produce long episodes of EEG sleep signs when sitting up as when lying. These experiments formed a sort of "control" for those of Section II. While perhaps some would disclaim surprise that, despite their obligations to remain alert, subjects went to sleep lying down, it was more remarkable that similar events occurred when subjects sat up and moved actively.

The quality of movement

When signs of sleep deepened, the skilled movements of pressing the switch disappeared before the cruder leg movements, though vague fumbling actions of the hand, in which the switch was held, might continue. In the case of the narcoleptic subject, leg movements often ceased as well, but would reappear momentarily before hand movements during a lightening of sleep, as judged by the EEG. Fig. 6 illustrates the disappearance of switch movement, and also shows the impairment of organization of leg movement.

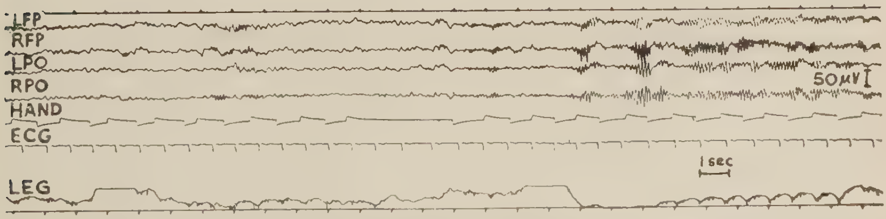


FIG. 6.—Subject A. The subject is pressing and releasing a spring-switch by hand and also moving his leg in time with the music.

The subject has drifted to light sleep and on the left can be seen disorganized leg movement which then improves when three bursts of low voltage alpha rhythm appear. Then sleep deepens again, the skilled movements of switch-pressing cease and the heart slows, but some leg movements continue crudely. Full alerting occurs on the right, with well-organized leg movement. An upward movement on the hand switch channel indicates a movement against the spring. The alpha rhythm tends to return after each such movement (cp. fig. 5).

Respiration

Synchronization of the rhythm of respiration with that of music and limb movement occurred to an extent which varied between subjects, and in the same subject, from time to time. Three subjects mainly were affected. The synchronization was irregular—there would, for instance, be a series of respirations at the rate of one per two limb movements, then a series of one per three movements, then a brief loss of any obvious relation, then perhaps a one to one synchronization. The synchronization was most marked when the music was very regular. When the rhythm became more difficult to follow, the respiration cycle tended to be prolonged.

While the influence on respiration was clear, it cannot be said that a tendency to cause actual hyperventilation was very obvious. Only two people showed a persistent tendency towards a one to one respiration/movement ratio. Subject G breathed deeply at the rate of 30 per minute for two and a half minutes, in the longest of several periods of one to one synchronization, and his respiratory excursion was deeper than normal during this period (fig. 7). After the session he complained of having experienced dizziness and sweating about three-quarters of the way through the session, which was the approximate time of this long hyperventilation. Subject H on the other hand, while showing similar synchronization, breathed only shallowly while doing so and stated afterwards that he had kept feeling the need to take a deep breath.

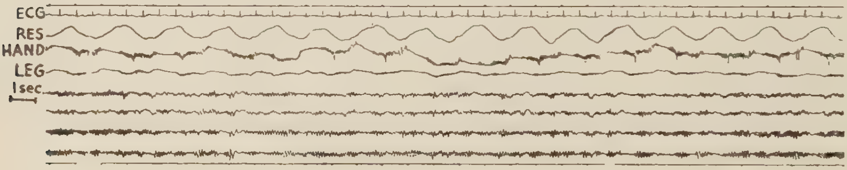


FIG. 7.—Subject G.
While this subject's respiration rate was mostly at 16 to 20/min. during movement to the music, several episodes of synchronization at faster rates occurred. A sample from one of these is shown.

The relatively staid laboratory conditions of the present studies were perhaps less potent in producing hyperventilation than would be the case where an individual was one of a group experiencing emotional excitement as a ceremony neared its climax. In the latter, too, the musical rhythms might be more regularly insistent, less obscured, than in the tape recordings. It could, too, be gradually quickened as a climax drew near. Furthermore, low blood sugar, and fatigue, enhance the EEG changes caused by the cerebral ischaemia that occurs with hyperventilation, and fasting and vigil are common precursors of certain ceremonies.

IV. General and Focal Cerebral Vigilance

Sleep and the EEG

Loomis *et al.* (1937), in the first account of a large-scale EEG study of sleep, stated that the stage of EEG flattening, in persons falling asleep, who normally showed alpha rhythm, was definitely to be regarded as sleep and as a stage in which dreams occur. Dement and Kleitman (1957) go so far as to claim that dreams are confined to this EEG stage. As sleep deepened, Loomis *et al.* reported, 4–6 c/sec. and 14 c/sec. waves were to be seen, and, later, slower waves of higher voltage. Their descriptions have been widely confirmed and accepted. Subsequently they and other colleagues noted that waves of alpha frequency might be lost at the occiput before the vertex, or vice versa (Davis *et al.*, 1938). Are we to conclude that part of the brain may be asleep while another part is not?

Pavlov (1955) wrote that "hypnosis", sleep, or internal inhibition, could sometimes be seen to spread gradually from involving first the tongue, then the jaw, and so on, of a dog, till gradually the whole animal was asleep. Are we to conclude that only part of the animal was initially asleep?

Roger *et al.* (1957) have reported a phenomenon observed also in the present study (to be discussed below), namely that during light sleep, EEG arousal signs related to a recurrent simple movement, may be confined to rolandic areas. Obviously the common understanding of what constitutes sleep does not fit easily within these facts.

Again, in the present experiments, when signs of sleep were seen in the experiments with stimuli at about every 10 seconds, no reasonable person could have doubted the presence of real sleep, for, apart from the EEG signs of flattening, slow waves and K-complexes, slowing of the heart and changes of respiration, there was unresponsiveness to sensory stimuli, occasional snoring, and dreams. Yet no clear distinction could be observed between the manifestations of sleep observed between the stimuli at 10-second intervals and those between stimuli at 5- and 3-second intervals or at the even faster dance rhythms. Miles (1929), from his study of eye movements, was of the opinion that one of his subjects, with eyes open, went to sleep, awoke, and went to sleep again, all within four seconds. Bjerner (1949) in an EEG study of performance at a continuous task in sleep-deprived subjects, and Magnussen (1944), from respiration studies, also described very brief moments of sleep. Sleep as a condition which might come and go every three seconds would not conform to what many understand by the term. Again, the EEG has indicated that there is no wakefulness–sleep dichotomy, but a wakefulness–sleep continuum. There is a need for a term which can more profitably be used to indicate changes from moment to moment of the physiological efficiency of large or small functional parts of the central nervous system. Head (1923, 1926) proposed that the term "vigilance" be used for just this purpose.

Vigilance

The degree of vigilance, Head maintained, indicated the degree of physiological efficiency. "Consciousness stands in the same relation to the vigilance of the higher centres as adapted and purposive reflexes to that of lower ranks in the neural hierarchy." When vigilance is lowered, "specific mental aptitudes die out", as if "the voltage falls below the necessary level"—the awareness of the subject of fig. 4 clearly fluctuated in this way. "The more highly differentiated the act, the greater the degree of vigilance does it require"—simple repetitive movements to music were still possible during signs of light sleep, but skilled movements became impaired—"as vigilance dies down, various forms of response disappear one by one according to the degree of neural potency necessary for their production". If there is "diminution of vigilance in any particular part of the nervous system . . . the loss of function may disturb certain aptitudes and not others".

Head wrote that vigilance could be lowered not only by sleep but by anoxia, chloroform and other agencies. The present study, it may be noted, has not only dealt with changes of vigilance of a sleep-like nature, but has indicated the potential lowering of cerebral vigilance by rhythmic stimulation, through hyperventilation and consequent cerebral ischaemia.

Contemporary knowledge has made possible the statement of Bremer (1954), of "cerebral vigilance being the condition of critical reactivity of the individual" which normally depends upon "continuous facilitatory dynamogenesis of cortical and diencephalic neuronal networks" by ascending impulses from the reticular formation of mid and hind brain.

If Head's recommendation be followed, no immediate objection to variations of vigilance, or efficiency, in different parts of the nervous system need arise. Again, we can speak of rapid fluctuations from moment to moment in the degree of vigilance, even at the rate of dance rhythms, or at the rate of the arterial pulse—evidence has elsewhere been presented to indicate that the inhibitory effect of baroceptor impulses on cortical function (Bonvallet *et al.*, 1954) may cause fluctuation of awareness with the pulse (Oswald, 1959a).

Changes of "rolandic vigilance"

The electrocorticographic studies of Gastaut (1952) and Penfield and Jasper (1954) emphasized the independence, in some persons, of occipital and rolandic electrical rhythms. They found that voluntary motor movement was associated with blocking of the rolandic rhythms. and that this blocking could be confined to the contralateral side, or even to, for instance, the motor hand area of the contralateral side. This blocking appeared before the onset of movement, and in different individuals could be effected by the instruction to be ready to move, or to imagine the movement.

Gastaut has emphasized that in some persons the principal rolandic rhythm seen in the EEG may be a rhythm at about 9 c/sec., with a characteristic non-sinusoidal, arch-shaped appearance, the "rythme en arceau rolandique". This blocks with movement or the thought of movement (Gastaut, 1952; Klass and Bickford, 1957), or following a signal which induces an expectancy of a subsequent signal for movement (Gastaut *et al.*, 1957, prefer to call this a cortical conditioned reflex). The rolandic rhythms tend to be unaffected by opening of the eyes, while the more posterior rhythms are blocked. Gastaut (1952), and Gastaut and Bert (1954), have pointed out a common manifestation of rolandic rhythms, namely that when a movement ends, the rolandic rhythms may return with an initial "rebound burst" of 18 c/sec. waves of perhaps accentuated voltage. This phenomenon is common, whereas the fully developed "arceau" rhythm is uncommon.

Gastaut relates these changes of rolandic rhythms to variations of "hyperexcitability" of the rolandic cortex and attempts to attribute them to disorders of the distribution of proprioceptive afferents from the latero-ventral thalamus and various other subcortical structures. These proprioceptive afferents are supposed to come from the part of the body involved, but Klass and Bickford (1957) report that "arceau" blocking occurred upon mental instigation of movement of a phantom limb.

It would seem more reasonable, at the present stage of knowledge, to suggest as a working hypothesis, that variations of the rolandic rhythm are related to "motor" attention, or vigilance. The "rebound" increase of the rolandic rhythm, after real or imagined movement, would be analogous to a similar "rebound" of the alpha rhythm, after it has been blocked, when there is a relaxing of attention. The burst of doubled frequency rolandic rhythm would be analogous to the doubled frequency, occipital rhythm, which, in many persons, briefly heralds the return of the alpha rhythm. This view is consistent with the claim that there may be unilateral enhancement of the "arceau" rhythm as a phenomenon of mild internal inhibition (Gastaut *et al.*, 1957).

Rolandic vigilance in the present studies

In the experiments of Section II in which subjects pressed a switch on hearing a faint tone, five of the subjects showed bursts of rolandic 18 c/sec. waves after pressing the switch, and in three of these, this rolandic activity was seen even though EEG signs of sleep persisted over the occiput. This phenomenon was also observed by Roger *et al.* (1957), but in the present experiments, since recording was bilateral, it was possible to see that in two of the subjects, the rolandic beta waves were occasionally contralateral as in fig. 8. It must be emphasized that usually occipital alerting accompanied rolandic, and that independence between these was the exception.

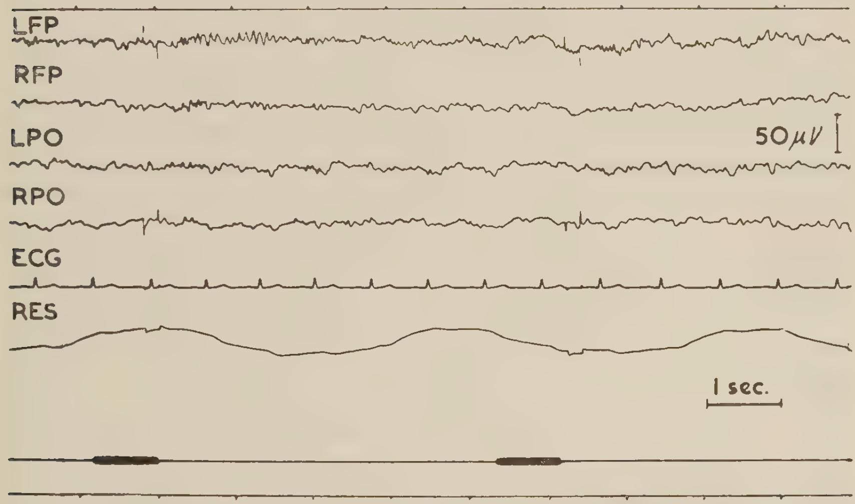


FIG. 8.—Subject A. The response marker, activated by the subject's right hand, is superimposed on the respiration channel.
The apparent phase-reversals of the switching artifacts are due to high resistance of the left frontal and right occipital electrodes.
Signs of sleep and alertness have been alternating at 5-second intervals but gradually sleep has deepened and the rolandic rhythms become more asymmetrical till, as may be seen, the subject is almost "asleep except for his left rolandic area".

It has been mentioned in Section III that the alpha rhythm sometimes came and went at the rhythm of movement to the jazz, as if the subject fell regularly into a light sleep. The alpha rhythm is particularly related to visual vigilance, however (Oswald, 1959b), and might not be expected to fluctuate consistently in an auditory-motor task. The rolandic rhythms are in a different category, and, in two subjects, showed very regular fluctuation with the rhythmic movements. The recordings from Subject D may be considered.

Firstly, in fig. 9, it can be seen that the usual burst of rolandic beta follows the tone when a response should have been made, but was not. It cannot be attributed in this case to proprioceptive afferents. It is quite clear also that the flatness of the record and the low voltage slow waves are

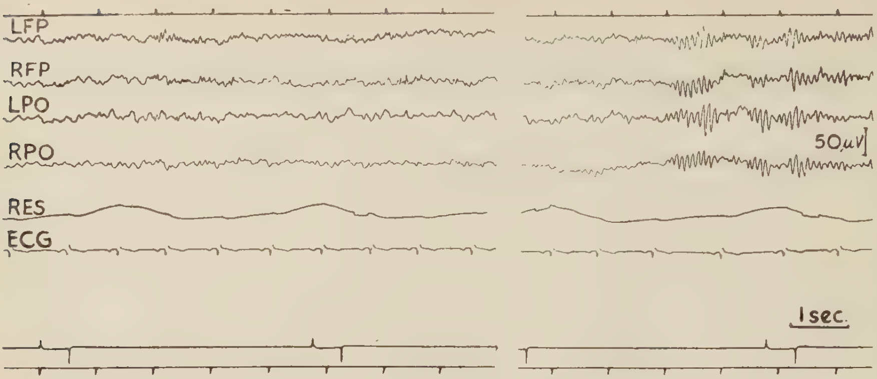


FIG. 9.—Subject D. The stimulus marker is shown at the bottom, above the time marker. The response marker is superimposed on the respiration channel.
Sleep has supervened but fast rhythms appear in both rolandic areas just after the response is made, or even (first stimulus) if the subject prepared to make it but did not actually do so. Shortly afterwards (right hand segment of illustration) a noise outside the room causes arousal and return to alpha rhythm.

associated with lowered vigilance or sleep; the noise causes a general alerting response.

When this subject sat up moving his hands and feet to music, major proportions of his records showed an absolute correspondence between the rolandic rhythms and limb movement (figs. 10 and 11). This was most easily seen during drowsiness, but could disappear if signs of still lower vigilance were present (figs. 10 and 12). The most striking feature in this subject was the appearance of bursts of bilateral 18 c/s waves of up to 50 or even 75 microvolts, which usually appeared just after the subject had banged down his hands and feet. This fast activity rapidly slowed to 10 c/s, and often to 4–6 c/s, during the "waiting period" in which the subject was slowly raising his arms and feet, preparatory to banging them down again. The relation of these fast rhythms to the immediately preceding downward bang of the hands

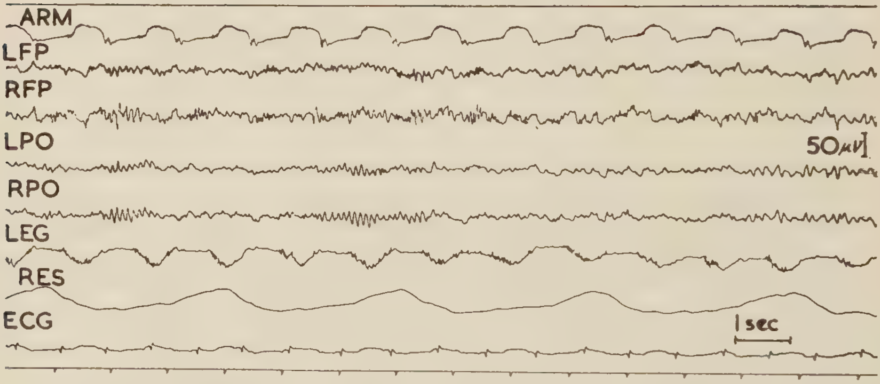


FIG. 10.—Subject D sitting up in a chair, moving his arms and legs to music. The subject is drowsing and alpha spindles are seen on the left, then the music changes to a quiet, sweet clarinet solo and sleep deepens with slow waves on the right.
Each downward bang of the hands every one and a quarter seconds is followed by a burst of rolandic fast waves, but these diminish as sleep deepens.

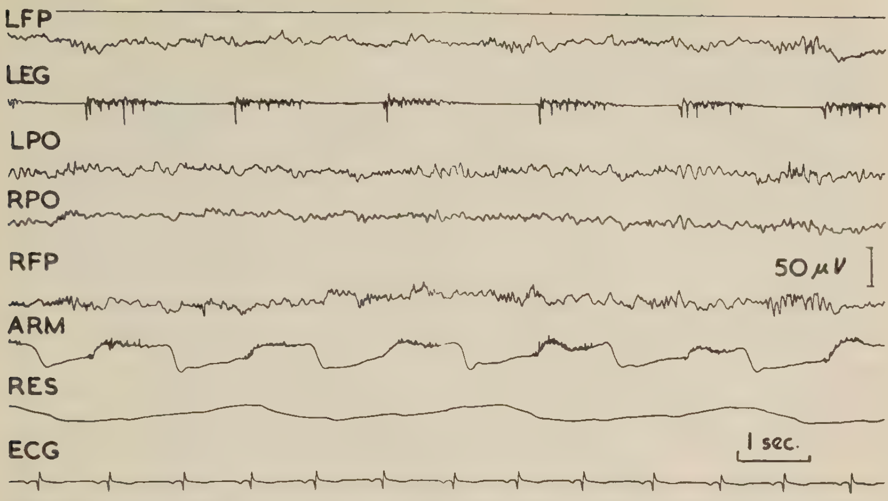


FIG. 11.—Subject D sitting up in a chair, moving his arms and legs to music. Relate the arm movement channel to the one above, from the right fronto-parietal area. The arm-channel shows muscle potentials on a rising curve as the arms rise slowly before being banged down (the sharp downward curve).
The subject has become sleepy again and while fast waves follow each bang, slow waves are seen while he waits to bang. Compare with fig. 9.

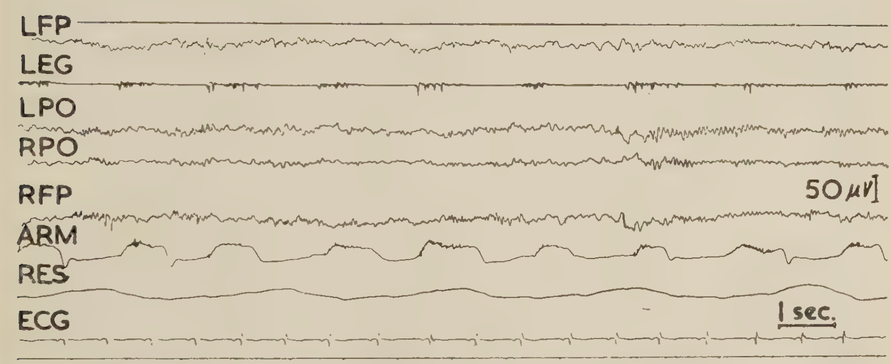


FIG. 12.—Subject D, as in fig. 11.
The subject drifts more deeply to sleep and the rolandic activity becomes disorganized, losing its usual relation to movement Arm and leg movements become suddenly small, fast rolandic activity does not replace the slow waves in the usual way, the subject is, as it were, "asleep all over" and then suddenly jerks awake (an artifact is visible in the left parieto-occipital channel). The bodily jerk was associated with an hallucination of something "flowing" across the body and was followed by 12 c/sec. EEG waves.

and feet, might be thought to support the theory that they are directly related to sensory input from the limbs. However, it was possible to see occasions in the record when these fast rhythms began just before the downward bang and continued during it (fig. 12). The appearance of beta waves after each bang bears a similarity to the appearance of alpha rhythm in fig. 5, for instance, where, following the movements and noises of the responses, bursts of alpha rhythm appear from a background of light sleep.

In figs. 10 and 11 there is alternation in the rolandic areas between a rather flat record with slow waves, on the one hand, and fast waves on the other. Could the flattening and the fast waves be equivalent to raised vigilance and relaxation respectively, as may be seen in an alert person with responsive rolandic rhythms? The answer must surely be, no. The flattening in the latter case is comparable to alpha blocking when vigilance is high (as when the eyes are opened). The flattening of figs. 10 and 11 is comparable to that of, for instance, fig. 9, where it represents light sleep. In fig. 12 "rolandic sleep" appears repeatedly in the waiting periods before the downward bang of the hands and feet, until the point is reached where, accompanying the low voltage slow waves, movement becomes impaired, and sleep is so predominant, vigilance so low, that movement is on the point of failing, at which point a "jerk" occurs, of the type which appears only when the individual has gone to sleep (Oswald, 1959c).

V. Discussion

The "harness" in "inhibitory experimental neuroses"

Sleep has often been described by animal experimenters, especially if the animal was repeatedly required to undertake tasks which were difficult for its limited capabilities (cp. listening for a faint tone in Section II). These "inhibitory experimental neuroses" have been seen in dogs (Pavlov, 1955; Dworkin, 1939; Anderson and Parmenter, 1941; Gantt, 1953), cats (Watson, 1954), rats (Cook, 1939), pigs (Lidell, 1944) and monkeys (Wendt, 1936), and in all cases the animals were subjected to physical restraint by some sort of harness. This applied also to the experiments of Yoshii *et al.* (1957), who made EEG recordings from a cat with an "inhibitory experimental neurosis", and observed sleep patterns. It must also have applied to a cat described by Jouvet and Hernández-Peón (1957) in which, if stimulus combinations of a tone and an electric shock were given "at a high rate" (three or four per minute), and if the shock was made strong, EEG signs of sleep were produced.

Wendt (1936) and Dworkin (1939) found that this reaction was replaced by more active behaviour, if freedom of movement was allowed, and Wendt was inclined to attribute the inhibitory state to physical restriction. However, it would seem that "psychological" restriction can be just as important. Liddell (1956) produced profound states of inert unresponsiveness in lonely lambs and kids, subjected to repeated mild electric shocks, and these had full freedom of movement within a room. Similar states produced by Masserman and Pechtel (1953) appear to have been related not to physical restriction, but to making monkeys, as it were, "feel hemmed in" by snakes. Again, physical restraint assists the production of "animal hypnosis" (see below), but it can be induced by "fascination" with a strong light in rabbits free to move, with production of EEG sleep signs (Gerebtzoff, 1941).

The subjects used in the experiments of Sections I and II not merely had a cap and electrodes on their heads, often electrodes strapped round their ankles or wrists, and a tube round their chests, but, above all, they accepted an obligation not to move, lest they disturbed the electrodes. The subjects in the experiments of Section III, though involved in quite active exercise, were under an obligation to make only certain movements.

This "harness" effect must be recognized as having played a possibly major role in the causation of sleep signs. It is, of course, obvious that individuals in daily life may feel hemmed in, or restricted, by obligations or dangers of real or delusional origin.

Monotony

Those who took part in the experiments of Sections II and III were involved in monotonous tasks. Monotony causes relaxation in man, this being true of, for instance, exposure to repeated noises (Lovell and Morgan, 1942) or even gum-chewing (Hollingworth, 1939; Freeman, 1940). The effects of monotony are related to the phenomenon of habituation.

Habituation

If stimuli repeatedly elicit a response from a nervous mechanism, then the response tends to decline or disappear. This is a basic property of nervous mechanisms of all degrees of complexity, being most rapid when "higher levels" are involved (Humphrey, 1933). Any novel stimulus tends to cause return of the habituated response to the original stimuli. Regularity of stimuli can impose environmental uniformity and this is a favourable, though not a necessary, condition for habituation (Oldfield, 1937).

In the present experiments, not only was habituation of the GSR seen, but also of EEG arousal, and a novel stimulus could restore the habituated arousal response (cf. the clap in fig. 2). In the experiments with electric shocks, habituation was manifest at the "highest level" as derealization. Similarly, Cohen (1954) described derealization and flatness of affect in Nazi concentration camp victims—they had been through so many horrors that they were now unmoved. Oldfield (1937) concluded that habituation does not occur to stimuli capable of damaging the organism. It would seem, however, that in the higher animals, including man, habituation can occur to dangerous stimuli, and that this may appear as a "change of standards" by which the danger is assessed. This change of standards may be forced on the organism after long-continued, but fruitless, responses, including anxiety, made to the danger, so that though the stimuli are still dangerous, they are no longer treated as if they were, so giving the impression to the observer of a "protective withdrawal from reality". The instances of derealization noted above are instances of this, and further examples will be given later.

Habituation in mammals can be accompanied by a degree of blockade of afferent inflow which, in the case of auditory stimuli, can take place at the level of the cochlear nucleus, and at the spinal cord in the case of somaesthetic stimuli (Jouvet and Hernández-Peón, 1957; Hernández-Peón *et al.*, 1956). In the present experiments, therefore, the effects of both the shocks and the auditory stimuli may have been modified in this way.

"Réaction d'assoupissement"

Gastaut *et al.* (1957) show the EEG of a person responding regularly to stimuli at 30-second intervals, and, drawing attention to the abruptness of the appearance of EEG slow waves, propose the term "réaction d'assoupissement" as a complement to "réaction d'éveil".

Now sleep tended to follow each period of attentiveness by those who listened for faint stimuli (Section II). Further, sleep or weariness is a sequel to a busy day, the effort required by an academic examination, or difficult car driving, and it can follow intense brief fear. Can it be that these events, and others mentioned by Sargant (see below), involving different time-scales, have something in common? The episodes of raised vigilance mentioned, all involve bombardment of the brain stem reticular formation by excitatory impulses. It can today be accepted that the level of activity of the latter largely controls the level of cerebral vigilance. Not only is the reticular formation stimulated by collateral afferents from all sensory paths, but also by impulses from higher centres subserving "voluntary attention", or attention following that appreciation of the significance of stimuli which higher centres make possible.

If, therefore, sleep, or lowered vigilance, occurs as a "rebound" phenomenon, does this in fact arise through a reactive depression of the activity of a reticular formation, which has become partially habituated to intense excitation, when that excitation ceases, through the ending of the stimulating situation, or because of the change of standards, or habituation at higher levels, mentioned above? Support is to be found in the studies of Hernández-Peón and Hagbarth (1955), who found that cortical electrical stimulation could be followed by a subsequent depression in reticular formation responsiveness to afferent stimuli. They proposed that this depression might account for the loss of consciousness following cortical epileptic discharges.

"Animal hypnosis" and its human counterparts

Not only is the altered responsiveness of habituation manifest throughout the animal kingdom, but so also, from cockroach to primate, is "animal hypnosis" (known by many other names; reviewed by Gilman and Marcuse, 1949). This condition of inert unresponsiveness can, like sleep in the present studies, be induced by rhythmic stimulation and bodily restraint. It can, however, also be induced by fear or gross physical stimulation, such as bodily rotation (which might be expected to involve fear and bodily restraint as well as a large sensory inflow).

Three independent EEG studies of this state agree that animal hypnosis is accompanied by EEG signs of sleep, which follow an initial picture of intense cortical activation (Gerebtzoff, 1941; Schwarz and Bickford, 1956; Svorad, 1957).

If intense, presumably terrifying, stimulation will cause animal hypnosis, will it produce a similar sleep-like state of inertia and unresponsiveness in man? There are many sources from outside the laboratory to indicate that this is so, though we may first, in passing, recall that anger, fear, sudden startle and laughter can cause human cataplectic attacks and that Adie (1926) and Wilson (1933) drew a parallel with animal hypnosis.

Wolfenstein (1957), reviewing some of the evidence, concludes that there is a "disaster syndrome" characterized by "absence of emotion, lack of response to present stimuli, inhibition of outward activity, docility and undemandingness". It is perhaps fair to liken the "docility and undemandingness" of these humans to the cataleptic maintenance of imposed postures seen in animal hypnosis and inhibitory experimental neuroses (Sidis, 1908; Foley, 1938; Anderson and Parmenter, 1941; Gantt, 1953).

Cohen (1953) described the reactions of prisoners entering Auschwitz concentration camp. Those who had repressed horrifying ideas before entry underwent "an acute fright reaction", which was "a state of stupor" which had "none of the somatic symptoms which are often observed in fright reactions". The long-term after-effects were apathy and depression.

Other descriptions of passivity, retardation, indifference to sensory stimuli and sleepiness may be found in descriptions of the victims of bombing (Harrison, 1941), "shell-shock" (Mott, 1919) and the survivors of a recent Atlantic liner disaster (Friedman and Linn, 1957). Numerous other examples are given by Sargant (1957), of stupor following intense emotion, and ether abreaction. He likens this to the stupor following electroplexy (cp. the previously mentioned suggestion of Hernández-Peón and Hagbarth).

Sargant shows, as has been suggested above, that the same type of reaction may appear to more protracted events, such as religious ceremonies, combat, psycho-analysis and "brain-washing".

Davis (1946, 1948), similarly described the reactions of bomber pilots to difficulty. Anxiety and overactivity sometimes gave way to inertia, apathy, dreaminess and unresponsiveness, while skilled movements became gross and disorganized (cp. fig. 6).

We have now reached a position where we can say that the lowered cerebral vigilance of the subjects exposed to anxiety, monotony and restraint in the present study, can be considered in a broader setting. We must recall the lowered vigilance found during movement to jazz, the potential lowering of cerebral vigilance through the ischaemia caused by hyperventilation, Sargant's emphasis on the effects of prolonged dancing, and that other agents he cites—intoxicant drugs, fasting and regulation of breathing—can all favour EEG slow waves. We can compare Wesley's converts that Sargant describes, who, after a period of mounting terror of hell-fire, when offered salvation, experienced new inner peace and glorious visions, to the subject of Section I, who, after prolonged and mounting anxiety, received relief, went to sleep oblivious of remaining stimuli, and experienced dreams of music.

Lowered cerebral vigilance is accompanied by a diminished awareness. A pre-requisite for the suggestibility Sargant believes is so manifest at these times is a diminished awareness of the nature of reality. Furthermore, the suggested beliefs can be learned or retained in a remarkable way. Many contemporary learning-theory psychologists have proposed that "drive-reduction", or tension-reduction, is an essential condition of successful learning. The new ideas the convert to Methodism or Communism accepted would achieve for him an enormous degree of "tension-reduction".

Hysteria and depression

The sleep of the subjects given electric shocks may prompt the comment, "Hysterical withdrawal!" Rivers (1922) considered human hysterical reactions to be modifications of animal hypnosis or the "instinct of immobility". Similarly Kretschmer (1926) thought them variations of the same "sham-death-reflex". Pavlov (1955) also interpreted hysteria in terms of internal inhibition, which, as pointed out, he identified with sleep.

Such views imply that an insoluble difficulty creates anxiety which is succeeded by a lowering of standards of achievement of "higher centres", and a decrease of cortico-fugal excitatory signals to the reticular formation. Thus would follow a reactive lowering of vigilance, confined only to certain functional groupings within the nervous system. Some support may be found in the report of Svorad (1957) that, in animal hypnosis, EEG slow waves may appear from some areas before others, that "motor" sleep or catalepsy is a precursor of the fully-developed flaccid, animal hypnosis (Sidis, 1908) and the evidence, from the present studies, of "focal" changes of cerebral vigilance.

Davis at first regarded the inert unresponsiveness of his subjects as hysterical, but subsequently as reactive depression in miniature (Davis, 1952), holding that the inertia and unresponsiveness of depressive illnesses could be attributed to habituation. Swank (1949) in an analysis of "combat exhaustion", described an initial "hyperactive" stage, with anxiety, and a second stage of "emotional exhaustion" with retardation, preoccupation, memory difficulties and apathy. Men who reached the second stage were "resigned to eventual death or mutilation". This is an example of the change of standards, the habituation to danger, previously discussed, where clearly a depressive state could be attributed to habituation. Daumézon and Lairy (1957) claimed that psychiatric patients who showed decreased reactivity to their surroundings had EEGs in which rhythms of wakefulness alternated with those of sleep, this being especially so in melancholic stupor.

Miscellaneous clinical phenomena

The continued movement associated with low cerebral vigilance in the jazz experiments, and the findings suggesting only "motor" arousal during more general sleep, are obviously reminiscent of sleep-walking.

The lapsing into sleep of subjects between electric shocks finds a parallel in the lapse into sleep of the parturient woman between labour pains.

The patient who lies immobile upon the psychoanalyst's dimly-lit couch makes recurrent responses, each associated in some way with an emotionally charged topic. One wonders to what extent each response reflects the dreams which, perhaps, filled an interval of sleep after the previous response. If the sleep deepens and long sleep-drifts appear, the analyst regards this as a sign of "resistance", and we have argued that a sleep-reaction is liable to be provoked by topics which evoke great anxiety.

Attacks of unconsciousness

It is very common, at least in young servicemen, for patients with anxiety-states to describe "black-outs". Many can be recognized as hyperventilation attacks. Other patients deny any similarity when deliberately asked to hyperventilate. Kennard *et al.* (1958) describe a study of 100 young people with behaviour disorders. Amongst these were a group who showed a special tendency to go to sleep during EEG recording, and this group had frequent bursts of frontal slow waves in their "waking" records. It seems possible, in view of the very brief episodes of lowered cerebral vigilance encountered in the present study, that the bursts of slow waves in the patients of Kennard *et al.*, and the "black-outs" of anxious patients may represent brief episodes of "sleep".

If one considers the instances of collapse after abreaction or threats of hell-fire, such as Sargant (1957) describes, they seem as much like swoons, or emotional faints, as sleepy stupors. The common swoon presumably occurs when inhibitory signals descend to the medullary reticular formation. Just as impulses from baroceptors not only inhibit the reticular formation of the medulla, but also that of the mesencephalon, causing sleep signs (Bonvallet *et al.*, 1954), may not also inhibitory impulses from the forebrain? While sometimes cardio-vascular changes may alone be sufficient cause of unconsciousness in carotid sinus syncope, in other cases the effects on the mid-brain may predominate (Ferris *et al.*, 1935). It is a reasonable conclusion that emotion might have a similar effect in certain swoons, causing "sleep" rather than cerebral ischaemia.

SUMMARY

Initially, a volunteer who had been subjected to very intense, irregular electric shocks, rapidly went to sleep one day when the shocks were less strong than he expected.

Further volunteers, who received regular, strong, electric shocks also went to sleep. Habituation of the galvanic skin response, and feelings of derealization occurred. Other subjects were subjected to the difficult task of listening for brief tones at the lower limit of audibility, occurring at regular intervals. Again there was an insistent tendency for sleep to appear over each session as a whole and for EEG sleep signs to occur between each stimulus-response, coming and going at, e.g. 3-second intervals.

Further experiments were carried out in which, for periods of an hour, subjects moved rhythmically to loud jazz music. Once again, EEG sleep signs appeared for long periods, even though movement continued. The quality of the latter tended to become impaired however. Once again EEG signs, as of fluctuation between alertness and sleep, at the rate of movement, were seen. Sleep appeared during movement to the loudest and most violent music, but most markedly when a sweet clarinet solo followed. Also when clapping and cheering helped the subject's escape to a dream-world.

In the experiments, the respiration tended to synchronize with the movement rhythm. Jazz rhythms occasionally caused hyperventilation. Characteristic differences between sleep and alertness were apparent in the shape of the respiratory curve.

Head's term "vigilance" was considered especially suitable to denote the varying cerebral states revealed by the EEG, especially when such variations were very rapid, or were localized to the rolandic areas, or even to the rolandic area contralateral to the hand in use. The hypothesis that the changes seen in these areas are related to proprioceptive afferents, was not supported.

The experimental results were related to those of Pavlov, to the phenomena of animal "inhibitory experimental neuroses" and "animal hypnosis", and to comparable human responses. It was argued that all the manifestations can be interpreted together in terms of current knowledge of sleep-wakefulness mechanisms and the phenomena of habituation.

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A PRELIMINARY EXPERIMENT ON PARANOID DELUSIONS

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THE ESSENTIAL REQUIREMENTS OF EXPERIMENTAL INVESTIGATION*

THE object of the experiment to be described in this paper, was to find out if it was possible to develop a systematic experimental investigation of paranoid delusions.

There are many psychologists who believe that it is not possible to apply, directly, the methods of natural science to the investigation of complex psychological disorders such as paranoid delusions. It is always easy to win a round of applause at a psychological meeting by asserting that the human mind is quite capable of comprehending, directly, its own subtle manifestations, and by asserting that the findings of experimental psychologists are too trivial to provide useful leads for the understanding of the more complex aspects of human behaviour.

This hostility to experimental method would not be supported by an impartial examination of the experimental literature. There are, however, some defects in the methods of experimental clinical psychologists, which provide some basis for this hostility.

These defects seem to arise from the fact that the essential requirements of experimental method have not been rigorously enough fulfilled. Paradoxically enough, these requirements would not be acceptable to the anti-experimentalist, though it is the failure to fulfil them which gives him some grounds for his dissatisfaction.

What are these requirements, and how do they affect the investigation of paranoid delusions? The first requirement is that of experimental control. In the case of paranoid patients this means that the method of enquiry should consist of the systematic changing of the situation of paranoid patients in such a way that the strength and quality of their delusions can be made to vary in accordance with these changes. Changes, both in the situation and in the delusions, should be clearly identifiable, and if possible, measurable. The situation which is being changed is usually called the "independent variable" and the phenomenon which is being altered as a consequence (in this case the delusions), is usually called the "dependent variable".

The second requirement of experimental method is that the experiments should have the aim of explanation. If possible, the changes in the independent variable should be those required by a particular explanation, and the consequent changes in the dependent variable, in this case the delusions, should be predictable from that explanation.

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* The first author is solely responsible for the methodological discussion and particularly for sections 1 to 5 of this paper.

The Choice of Phenomenon for Investigation

The research work of experimental clinical psychologists usually fails to fulfil these requirements in at least one of three ways. The first kind of failure consists of the fact that the phenomena which are chosen for investigation appear, at first sight, to have little relationship to the disorders which bring psychiatric patients into hospital. Psychiatric patients suffer from a variety of disorders of affect, cognition, and volition. A large number, if not the majority, of papers published by psychologists do not deal with these phenomena. Instead they report upon the performance, by psychiatric patients, of a variety of tasks which might be described, without much loss of accuracy, as puzzles and indoor games. Examples are such tests as the pursuit rotor, the mirror drawing test, the block design test, the Rorschach Test and the thematic apperception test, and the rotation effect (13). For purposes of discussion this approach will be referred to as the *indirect approach*. If we were to apply the indirect approach to the problem of paranoid delusions, we would have to find some task which at least some paranoids did differently from all other subjects, including normals and psychiatric patients. Having succeeded in finding such a task, we would proceed to investigate it.

Several arguments are advanced in defence of the indirect approach. The first argument is that the scientist must take as his starting point those forms of behaviour which are amenable to experimentation. Such an argument would really only hold water in the investigation of delusions if a direct experimental attack upon them had failed. To our knowledge, no such an attack has ever been made.

The second argument which is put forward in favour of the indirect approach is that the scientist can deal with only a limited aspect of any phenomenon which is under investigation, and that the findings which emerge should be used to throw light on other and perhaps more important aspects of the same phenomenon. This argument has force in so far as it implies that the experimentalist might be restricted at first to the investigation of those manifestations of delusions which are more easily measurable. This does not mean, however, that the psychologist is forced to investigate some quite different function like, for example, mirror drawings. He is only forced to do the latter if he has failed in his attempt at the direct experimental manipulation of delusions, or if he is, in fact, more interested in the psychomotor functions involved in mirror drawing than he is in delusions.

A third argument advanced in favour of the indirect approach is that the investigation of any single function is bound to throw light on other functions. If paranoids have a way of doing a puzzle which is peculiar to them, then the investigation of the way they do this puzzle may well throw light on the nature of paranoid delusions. Admitting that this might be the case, it would still be necessary to devise experiments on paranoid delusions directly, before we could accept the theories which had been suggested by the indirect investigation. It would, therefore, seem to be more reasonable if it is desired to investigate paranoid delusions, to start by trying to investigate some direct manifestations of these delusions.

Finally, it should be pointed out that the indirect approach has a general disadvantage. Successful research depends on close attention to detail, upon knowing a tremendous amount about all the factors affecting the appearance of the phenomena under investigation. If one uses, say, a perceptual test to investigate deluded patients, one would have to learn a great deal about perception in general, and the particular aspect of it which one was investigating. In the meantime one would have learnt very little about the nature of delusions, and, therefore, it would be very difficult to make discoveries of an inductive character. A method of work which restricts the possibilities of systematic induction would seem to be faulty.

The direct systematic investigation of some aspect of delusions might at least lead to some generalizations of limited applicability, and of some practical use in the management of patients who suffer from them.

In view of all these considerations, it was decided to try to investigate some direct manifestation of delusions.

THE CHOICE OF METHODS OF INVESTIGATION

The second way in which experimental clinical psychologists often fail to fulfil the essential requirements of experimental investigation is in their choice of methods of research. They tend to concentrate upon the statistical description and manipulation rather than on experimental manipulation. Current procedures often consist of obtaining a large number of test scores from various nosological groups. The psychologist then determines the way in which these test scores differentiate between groups and the way in which they intercorrelate.

The dependent-independent variable relationship is, of course, a kind of correlation. The essence of the method of experimental control, however, is that the phenomenon under investigation is made to change in a given individual or group in accordance with controlled changes in circumstances. In this way causal elements can be more unequivocally identified, and processes are more directly observed.

THE ATTITUDE TO THEORY

The third way in which the experimental clinical psychologist tends not to fulfil the basic requirements of experimental method, is in his attitude to theory. This is exemplified especially by those psychologists who express allegiance to various forms of the hypothetico-deductive method. Their work is directed mainly at proving or disproving certain theories rather than at explaining and controlling certain phenomena which have excited their interest. In fact it sometimes seems as if some psychologists look upon experimental method merely as the most convincing method of persuading people to accept their ideas. It thus ceases to become a method of determining the actual state of affairs, independently of a priori conceptions.

Actually, the main advances in biological science seem to come from men with a different approach. Darwin was mainly interested in co-adaptation. An example of co-adaptation is the fact that the "feet, tail, beak, and tongue of the woodpecker are so admirably adapted to catch insects under the bark of trees" (2, p. 32). Sherrington was interested in the fact that the spinal nervous system behaved in an apparently purposive and adaptive manner, and Pavlov in the fact that the higher nervous centres showed the ability to make and unmake connections with relative ease. The generalizations they developed arose mainly from the results of the active manipulation of the phenomena which interested them.

In developing an explanation of delusions we would, if we were "theory-oriented", write down all the theories which important people had uttered about delusions and proceed to do experiments to test them. We would not care much about the kind of phenomena we observed in doing so, so long as they lent themselves to our purpose. Unfortunately, as far as we have been able to gather, all the current "explanations" of delusions are related only to observations of an anecdotal character. The theories then have limited immediate scientific value. Even the supposed most important characteristic of the delusions, the relative lack of modifiability, has, as far as we have been able to ascertain, been described only in an anecdotal manner. We could find no record, for instance, of someone having tried to "talk a patient out of" a delusion over a definite number of hours, using a certain approach and certain measures of the strength of the delusions concerned. Not only are the available theories of limited usefulness; but we have no experimental observations of the phenomena concerned.

THE NEED FOR A BALANCED APPROACH

It is not being argued here that the indirect approach, or the studies of individual and group differences, or the interest in theory, are all useless. The facts clearly indicate the contrary. For example, it is of considerable interest that schizophrenics, in two out of three independent experiments, have been found to produce errors in their judgment of size which deviate more from "retinal" size than do the judgments of normals (4, 6, 7). Likewise the United States Employment Service (16), in arriving at a relatively stable factorial description of human ability, helps us make a relatively organized and streamlined description of the cognitive efficiency of an individual, and sets clear problems for further research into the nature of human ability. Finally, Hull's attempts to formulate a theoretical system have stimulated a considerable amount of systematic research into the nature of the learning processes. In fact, any method of systematic observation of behaviour is likely to be useful, and might even result in important discoveries. All that is being argued here is that there is a dearth of direct experimental enquiries into the nature of psychological abnormalities. It is time that this deficiency was corrected.

With all these considerations in mind we turned to the investigation of delusions. We decided to carry out our first study on a single paranoid patient. We have discussed elsewhere the methodology of the single case (11). All we can say here is that there is some evidence that the single case investigation is an economic and speedy method of opening up a new field. The senior author and various colleagues have developed three lines of investigation on psychiatric patients in this way: one on the block design rotation effect (9, 10), one on slowness of psychological function (12), and one on reading disability (1). An additional reason for using the single case investigation is that we were interested in trying to develop methods combining clinical and fundamental research.

THE SUBJECT

The subject on whom we decided to carry out our first investigation was a thirty-eight year old male who was diagnosed as a paranoid schizophrenic. As a young man he had been studious and interested in amateur dramatics. During the second world war he had been a pacifist and a conscientious objector. Since then he had maintained a preference for the "simple life" on a farm.

He had worked as an agricultural labourer without advancement. His Full Scale Wechsler I.Q. was 126, with a Verbal I.Q. of 130 and a Performance I.Q. of 118. In conversation he gave the impression of being extremely economic and clear in self expression. During his stay in hospital he experienced an acute depression which cleared up after 8 doses of E.C.T. administered over a period of 41 days. The patient's paranoid illness appeared to have been of about five years' duration.

The patient's main delusions consisted of the belief that there was an association of people who were trying to change him and punish him. They were doing so, he believed, on account of his defects. These defects were his pacifism during the last war, his sexual misdemeanours which he reported as consisting of a few homosexual episodes, six episodes of bestiality, and his sexual and moral inadequacy as a husband.

The patient had not, for 21 years at least, been a member of any political organization. He was an ordinary citizen whose life was taken up by his work and by his family.

His "persecutors" were watching him constantly, either by direct surveillance or by microphones placed in his home. They had immediate knowledge of the most intimate details of his behaviour, and could possibly influence his thoughts.

In addition to these beliefs about the "organization" the patient believed that his wife had been unfaithful to him, with the result that his five children were not his own. He believed that he himself was illegitimate. The patient appeared to be suffering from no other psychological abnormalities.

FORMULATION OF THE PROBLEM

This was based on the outcome of discussions held with the patient by Mr. Patten.

The following statements could be made about the patient's beliefs. Firstly, the patient could be said to have a number of palpably false beliefs of a paranoid character. For example, in our society, there are no groups of politicians aiming at obtaining political power through the modification and punishment of ordinary citizens like our patient. Not all of the patient's beliefs which gave him cause for concern were palpably false, e.g. the belief in his wife's unfaithfulness.

Secondly, it appeared that the paranoïd beliefs were abnormally resistant to the usual processes of modification.

Thirdly, it could be argued that the paranoid beliefs were not in the interests of the patient or of society, because they might result in his committing acts of violence against his family and himself, in his relationships with his fellow men being ruined, and in giving him unnecessary misery.

This formulation does not imply that there might not be people, outside the mental hospital, who have beliefs concerning which the same three observations could not be made. We were mainly concerned with this particular individual as we found him, and those features of his condition which appeared to require help, and hence merited research by the clinical psychologist.

The statement that the patient's paranoid beliefs were abnormally resistant to modification is, as we stated above, a statement that required confirmation. It seemed therefore that, at the risk of looking foolish, we had to begin with the systematic observation of the reaction of the patient's beliefs to such processes of modification. The process which could be provided by us most easily was the process of discussion. It was, therefore, decided to hold a series of discussions with the patient.

General Plan of the Investigation

In planning these discussions we at first made an attempt to deduce, from some of the usual explanations of paranoid delusions, the kind of discussion which should be held. For the reasons stated above we did not pursue this approach too rigorously. Our final plan of discussion, therefore, was arrived at somewhat arbitrarily. First of all, we had to have some essentially rational discussions aimed at demonstrating to the patient the palpable falseness of his persecutory delusions, and aimed at supplying him with possible explanations of how he might have come to have such delusions. Secondly, we thought it necessary to have, as a control, discussions of another kind. For this purpose we decided to have discussions of guilt, with the aim of modifying guilt feelings where they appeared to be unwarranted, and of facilitating the handling of the issues which arose. We were quite aware that the guilt feelings might also have a delusional quality, though this was not as yet as clear to us as the delusional quality of the persecutory beliefs.

Because the patient's length of stay in hospital was uncertain, we decided to have as many discussions as we could manage per week. Neither of the authors had the time to do this alone, so it was decided to involve both of the authors in the discussions.

In these decisions we were operating the independent-dependent variable model. The independent variable consisted of the discussion produced by the two authors. As part of the independent variables we were ourselves reproducible in so far as we were available to take part in further experiments. This reproducibility was limited by the possibility that our experience of the discussion might change our behaviour. Ideally, we, as discussers, should have been described in quantitative terms, so that other discussers similar to, or definably different from, ourselves could be found for further experiments. We should have been described in terms of our impact on other people and particularly upon the patient. We felt, however, that we knew too little to begin this kind of experimentation. We felt it was better to go into a period of relatively uncontrolled observation of both the patient and ourselves in this kind of discussion before trying to quantify ourselves.

For similar reasons we decided not to lay down rules about how the discussion should be tackled and what topics should be raised. It was left to each experimenter to conduct the discussion in the manner he thought best. The only restrictions were related to (*i*) the topic, which was either that of guilt or paranoid delusions, and (*ii*) the commitment to discuss these topics on as rational grounds as we could muster. All one could say, therefore, is that the two discussers provided two complex forms of stimulation, the content of which could be identified as the rational and humanist approach to problems arising from intense guilt and from palpably false beliefs about the actions of other people. Both of the discussers were verbally fluent people who normally enjoyed a rational and friendly argument.

The dependent variable presented an easier problem. One of the main ways of finding out about a subject's beliefs and feelings is to ask him about them. It was decided, therefore, to use a questionnaire method. This decision is justified by an examination of the relevant literature which indicates that answers to questionnaires have some relationship to the variables which they appear to measure. This statement is exemplified by findings which show consistent and appropriate relationships between questionnaire responses and the following groups of observations:

(*i*) the diagnosis of neurosis and schizophrenia (17)
(*ii*) psychological functions such as perceptual threshold and memory efficiency (15)
(*iii*) physical conditions like gastric ulcer (16)
(*iv*) concrete behaviour such as delinquency (3).

It was decided to use four questionnaires. The first was to measure paranoid belief, the second to measure guilt, the third hostility, and the fourth depression. The depressive questionnaire was decided on because Mr. Patten noticed in his interviews that the strength of the patient's paranoid beliefs and his depression appeared to vary concomitantly. The hostility questionnaire was added because of the current notion concerning the relation of hostility and paranoia. The four questionnaires are given in Table I.

TABLE I

The Questionnaire

		Score on First Occasion	*Score on Last Occasion
Depression (*D*)			
1	It is harder for me to be cheerful than it is for most people	1	0
2	I find it difficult to have any sort of pleasurable feeling	1	0
3	I feel I have a heavy burden to bear in life	0	0
4	I sometimes feel dead inside	0	0
5	On the whole I do not look forward to the future with pleasure	4	0
6	I feel that life for me is full of misery, pain, hardship and very little besides	1	0
7	I can't seem to get any satisfaction out of the things that I do	1	0
8	I feel now that life is just not worth living	0	0
9	I feel so depressed that concentration is difficult	3	0
10	I feel miserable today	0	0
Guilt (*G*)			
1	I feel that I am an unworthy man	4	4
2	I have every reason to feel guilty	4	4
3	I feel that some of the things that go wrong are my own fault	4	4
4	I experience strong pangs of conscience	4	4
5	People would despise me if they really knew me	4	4
6	I have the feeling that some part of me is wicked	4	4
7	I feel I am a bad person	3	4
8	I feel remorse when I think of some of the things I have done	4	4
9	I believe that my sins will find me out	4	4
10	I feel ashamed of myself	4	4
Hostility (*H*)			
1	I find myself irritable without any real cause	0	0
2	I find myself aggressive without any real reason	0	0
3	People seem to try to pick quarrels with me	1	0
4	I get a sneaking feeling of pleasure when people I dislike get into trouble	1	0
5	I carry a "chip on my shoulder"	0	0
6	I am very touchy	3	0
7	Unimportant things irritate me	3	0
8	I am impatient with others	3	0
9	I feel I get a raw deal out of life	1	0
10	"Never trust strangers" is my motto	0	0

* This is the last occasion of the actual experiment.

Table 1—continued

		Score on First Occasion	*Score on Last Occasion
Paranoid Beliefs			
1†	I believe that a minority group is secretly organizing a campaign against me	1	0
2	I believe "they" could be more powerful than any government or nation in the world	4	3
3	I believe "they" want to influence me	3	3
4	I believe "they" use microphones and electronic devices to keep a record of my every move	3	3
5	I believe "they" observe me everywhere I go	4	3
6	I believe "they" will keep watch over me in the future	4	3
7	I believe that "they" are trying to retard the growth of my children	3	2
8	I believe that "they" may be working to further eugenic ends, e.g. to eliminate an undesirable strain	2	3
9	I believe "they" are using methods of non-violence	4	3
10†	I believe that by a subtle use of the law "they" have put me "inside"	4	0
11	I believe that I cannot escape from "them"	4	3
12	I believe that I am not the true father of my children	2	0
13	I believe that my wife has been unfaithful to me	2	0
14	I believe that I am not the son of my supposed parents	2	2
15	I believe that one reason for "them" getting at me is my pacifism	4	3
16	I believe that one reason for "them" getting at me is my failure as a man	4	3
17	I believe that one reason for "them" getting at me is my failure as a husband and father	4	3
18	I believe that my children are physically retarded	2	2
19	I believe that I have physical abnormalities	3	2
20	I believe that dope has been put into my food to make me irritable	3	0

* This is the last occasion of the actual experiment.
† These items were eliminated from the statistical analysis.

The items for the paranoid questionnaire were made up specially on the basis of Mr. Patten's interviews with the patient. The remaining items were drawn from Sandler's questionnaire (8) but modified for use with a five point scale.

To sum up, we had the following main questions:

(*i*) Would the rational discussion of paranoid delusions make any difference to their strength?

(*ii*) Would the rational discussion of beliefs of guilt make any difference to the strength of delusions?

(*iii*) Would changes in the strength of paranoid beliefs be related to changes in the strength of feelings of guilt, depression and hostility?

PLAN OF INVESTIGATION

The procedure operated is illustrated in Table II. An attempt was made to balance, in relation to other commitments of the two authors, the discussion of guilt and paranoid beliefs. The questionnaires were administered once per interview, half of the time before, and half after each interview. The aim of this balanced order was to enable us to get some kind of measure of the actual effect of the interview itself. The questionnaires measuring paranoid belief, guilt, and depression were shown to the patient before the experiment began.

He accepted them as being appropriate and understandable. The hostility items were, by accident, not shown to him.

Sixteen interviews, each of about one hour's duration, were held at the rate of four a week, on Monday, Tuesday, Wednesday and Thursday of each week. The first two interviews were held on a Wednesday and Thursday. It was felt that if this amount of work produced no changes of interest, then we would have to reconsider the value of this approach. These decisions were based on practical considerations only.

A method of exact recording was not used. The authors took brief notes during each interview, and immediately afterwards wrote a summary of what they thought had transpired. The authors have very little confidence in the value of these reports. A summary of the arguments presented to the patient are, however, given below.

Each question was written on an index card 5 inches by 3 inches, resulting in a pack of 50 cards. These were shuffled before each interview, and the subject was required to place each card under one of five headings: "This is definitely the case", "This may be the case", "I am not sure", "This may not be the case", and "This is definitely not the case". For purposes of quantitative analysis, to the first heading was arbitrarily assigned a score of 4, the second 3, the third 2, the fourth 1 and the last 0.

On the whole the patient appeared to be very co-operative and glad to talk. Sometimes the pressure of his talk was so great that it was difficult to break off the interview. The demeanour of the patient appeared to change over the course of the interviews. At the beginning he appeared to be positively hostile. In fact, as he admitted later, he was on the verge of assaulting the psychologist on two occasions, each author being once threatened in this way.

Dr. Mallett, the registrar in charge of the case, decided to continue to see the patient twice a week throughout the experiment. In these interviews the registrar continued to take his usual neutral attitude to the delusions and avoided associating himself directly with the experiment being carried out by the psychologists.

SUMMARY OF THE CONTENT OF THE ARGUMENTS PRESENTED TO THE PATIENT

(i) *Paranoid Beliefs*

There were three aims in these discussions. The first was to demonstrate the palpable falsehood of the patient's paranoid delusions whenever the opportunity offered. The second aim was to prove to the patient that his entertainment of such false beliefs was a proof of illness. The third aim was to show that it is possible to advance explanations of how an intelligent man could come to have such an illness. It was, of course, made clear that no such explanations could, at the moment, be proved.

Three examples of our approach to specific paranoid delusions must suffice. The belief that a political organization was persecuting him was tackled by pointing out that a serious political organization in Great Britain would want to gain political power, and that it would be a ludicrous waste of time for such an organization to involve itself in the elaborate spying upon and persecution of an ordinary individual.

The belief that he was being watched was shown to the patient as not being supported by a single clear incident which he could recall. The belief was then reduced to the fact that he *felt* he was being watched, a feeling which need not necessarily have a real cause to be experienced.

His belief that his children were not his own was discussed by pointing out that each of his five children had been born in different parts of England and over a period of 18 years. This probably meant that a number of men would have had to be involved. Despite this possibility, the patient had never seen anything or been told anything that could plausibly be regarded as evidence of his wife's unfaithfulness.

The more general approach consisted of our initiating discussions on such topics as the relation between belief and impression. For example, the patient seemed to be impressed by the argument that interpretation of "appearances" could be affected by previous belief, and these appearances could in turn act as reinforcers for such beliefs. The attitude of the authorities in the 15th century to the first statements of the Copernican theory was given as an example.

Another example of this kind of discussion was the suggestion that the patient might in effect be having hallucinations of belief. Experimental work on sensory hallucinations was quoted, especially that of Penfield on the direct stimulation of the exposed cortex. It was then suggested that the spontaneous excitation of the arrangements underlying the verbal system might produce "hallucinations" of belief which, it was suggested, any man would find it difficult to withstand. This explanation was related to his own reported experiences which were analogous to the primary delusions described by Mayer-Gross, Slater and Roth (5). Discussion of this kind was, of course, undertaken in terms which the patient could understand.

(ii) *Guilt*

As with persecutory delusions, guilt was attacked both specifically and generally. One of the specific points discussed was the patient's pacifism. It was pointed out that pacifism probably created a dilemma for him in that, while his community was fighting for its existence he had been doing nothing, yet at the same time he had accepted the benefits from that community, and from the successful prosecution of the war. Nevertheless, in standing by his beliefs against the current of opinion, he had shown moral qualities of which he could be proud, and which were valued by many individuals who did not in fact share his views.

A second example was the guilt he felt over his emotional outbursts. It was argued that these were aspects of behaviour which might be open to explanation and possible modification.

The main general issue discussed with the patient was the impact upon the individual of the conflict between necessary and rational requirements of society on the one hand, and forceful vagaries of human nature on the other. The psychologists maintained that it was necessary for man to combine an aspiration towards the highest personal standards with an understanding of his own and other people's failures in reaching them. The formulation which appeared to have the most force with the patient was that there was a difference between the forgiveness and the condoning of an offence. The senior author occasionally experienced some difficulty in discussing guilt because of his own feelings that the patient's feelings of guilt were on the whole quite justified on the evidence as presented.

QUANTITATIVE RESULTS

(*i*) AFFECT SCALES

(a) *Depression and Hostility*

On the basis of our arbitrary scoring system the maximum score possible for the ten Depression Items and ten Hostility Items was, in each case, 40 points. Superficially the scores, as tabulated in Table II, suggest a progressive reduction and eventual disappearance of depression and hostility. These results cannot, however, be accepted at face value. We took the precaution, at the end of the experiment, of discussing each of the items of the questionnaires with the patient. We found that six of the depression items and seven of the hostility items had been rejected by the patient because he had come to believe that the wording was inappropriate. He therefore rejected them though he would have accepted them if the wording had been slightly changed. For example he rejected the Depression Item 8 "I feel now that life is not worth living", because he thought that the word "existence" should have been substituted for the word "life". If that had been done he would have sorted the item under the heading "definitely the case". It seemed to us that the patient, despite his high intelligence and apparent lack of impairment on formal tests of thought disorder, had some difficulties of thinking when the material was concerned with depression and hostility. We did not have the time to investigate this observation, so there is no point in discussing the matter further in this paper. It was clearly impossible to draw any conclusions, from this material, about his fluctuations in hostility and depression. We therefore made no further analysis of the data.

TABLE II
Main Results

Content of Discussion	Guilt				Belief								Guilt				No Discussion		Guilt	
Interviewer	R		S		R		S		S		R		S		R		R	S	S	S
Before/After interview	B	A	A	B	B	A	A	B	B	A	A	B	A	B	B	A				
Depression	11	19	11	15	10	9	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Guilt	39	36	32	40	40	36	40	40	36	40	40	40	38	38	36	40	40	40	36	35
Hostility	12	14	16	10	7	8	6	4	3	9	9	6	0	0	0	0	8	3	9	9
Paranoid*	57	46	52	54	54	53	48	50	50	47	39	41	40	40	44	41	37	43	39	42
Day of the week ..	W	Th	M	T	W	Th	M	T	W	Th	M	T	W	Th	M	T	1+	2++	3+++	

R = Ravenette S = Shapiro 1+ 14 days later 2++ 36 days later 3+++ 46 and 47 days later
* Based on 18 items Total scores for each scale for each occasion

(b) *Guilt*

The maximum score possible for the ten items was 40 points. In contrast to the hostility and depression scales, no items on the Guilt Scale were rejected in our final discussion with the patient. An examination of Table II and of the graph will show that these fluctuations were not very large, though, on the whole, the strength of guilt was deemed to be weaker during the guilt discussions than during the discussions of persecutory beliefs. An analysis of variance was carried out which gave an F ratio of 3·28. An F ratio of 3·92 is necessary if the 5 per cent. level of significance is to be reached. We can only conclude that there might have been a small but real weakening of the feelings of guilt in response to the discussion of guilt.

(*ii*) PARANOID SCALES

Twenty items had been initially made up for these scales. In the last interview only two of these were rejected by the patient on grounds similar to those which caused the rejection of the depression and hostility scales. We were thus left with eighteen items for which the possible maximum total score was 72 points.

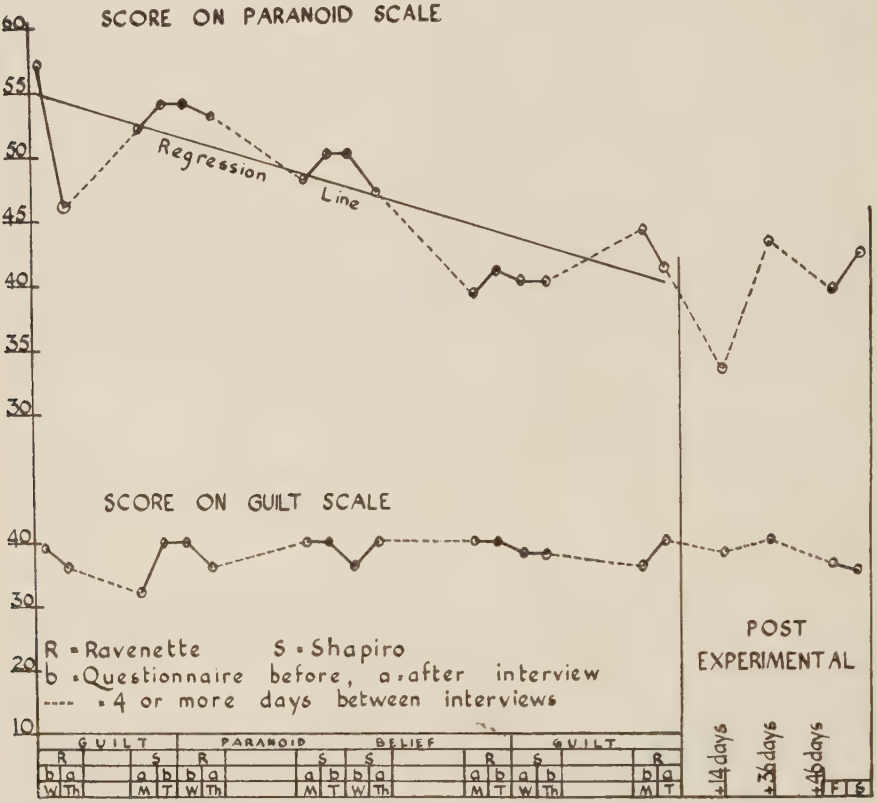

FIG. 1.—Changes in Paranoid and Guilt Scores.

An examination of Table I and of the Graph shows that there is an apparent fall in the strength of the paranoid delusions with an occasional fluctuation. An analysis of variance was carried out which resulted in the differences between the 16 occasions giving an F ratio of 5·74 which is significant at beyond the ·001 level. This result indicates that the downward trend is probably significant. The difference between items gave an F ratio of 24·82 which is also significant at beyond the ·001 level. This finding indicates that though the total scores for each of the occasions differed significantly from each other, the *relative* strength of belief for each of the 18 items tended to remain the same on all occasions, i.e. the patient showed relative consistency of response (see Table III).

TABLE III

Overall Analysis of Variance

Source	d.f.	S of S	M.S.	F Ratio	P
Between items	17	139·875	8·288	24·82	<·001
Between occasions	15	28·944	1·929	5·74	<·001
Residual (error)	255	85·181	·334		
Total	287	253·500			

To check the downward trend more precisely, a regression line was fitted, giving the regression of paranoid delusional strength upon occasions. This line is shown as a straight line in the Graph. The variance accounted for in this way gave an F of 19·3 which was significant at beyond the ·001 level (see Table IV). The residual variance was, however, significant between the ·01 and ·05 level. This implies that the fluctuations in delusional strength around the regression line are still significant.

TABLE IV

Analysis of Variance Assuming One Regression Line

Source	d.f.	S of S	M.S.	F Ratio	P
Accounted for by regression	1	19·336	19·336		<·001
Residual variance for occasions	14	9·608	·686	2·055	0·1<P <·05
Total variance between occasions*	15	28·944			
Residual (error)*	255	85·181	·334		

* From Table II.

(It should be pointed out here that regression lines on these data are not statistically beyond reproach. Regression on time implies equal units of time but with our data "occasions" are not separated by equal units of time.)

Two explanations of these fluctuations can be advanced. The first is that the discussion of paranoia had a more powerful influence than the discussion of guilt, as an inspection of the Graph seemed to show. To check this suggestion, we carried out an analysis of variance in which it was assumed that no changes occurred during the discussion of guilt, and that the only changes occurred during the discussion of delusions. In effect two straight horizontal lines were drawn, one for each of the two periods of guilt discussion, and one regression line to cover the period of discussion of paranoia. The residual variance, as consultation of Table IV will show, was significant between the ·10 and the ·05 level. We, therefore, could not be satisfied that this hypothesis fully accounted for the fluctuations.

The second explanation, suggested by Mr. P. Slater, was that all changes, for the worse or the better, only occur during the week-ends. An examination of the graph will show that the four questionnaire measures during each of the three groups of midweek discussions, produce an approximate plateau every time. The largest changes, except one, took place over the week-ends. An analysis of variance (see Table V) was then carried out in which this pattern was assumed, there being straight horizontal lines joining each set of midweek discussions. In addition, straight lines were drawn for the first two and the last two discussions. The residual variance failed to reach significance at the ·20 level. The second explanation therefore accounts for the fluctuations more efficiently than the first. The design of the experiment does not, however, enable us to make a clear choice between the two explanations. It should be noted, as inspection of the graph will show, that the paranoid delusions became *stronger* in the week-ends succeeding the discussion of guilt, and *weaker* on those week-ends succeeding the discussion of delusions. This interpretation is given added interest by the observation, reported above, that there was a slight tendency for the feelings of guilt to be weakened by the discussion of guilt, though that tendency did not quite reach an acceptable level of significance.

TABLE V

Analysis of Variance Assuming: (1) *All Change Occurs at Week-Ends;* (2) *All Change Occurs During Belief Discussions*

Source	d.f.	S of S	M.S.	F Ratio	P
Total variance between week-ends ..	4	24·694			
Residual variance between occasions ..	11	4·250	·3864	1·157	P> ·20
Due to mean differences, Weeks 1 and 4, and regression in Weeks 2 and 3 ..	4	22·561			
Residual variance between occasions ..	11	6·383	·5803	1·737	·10>P > ·05
Total variance between occasions* ..	15	28·944			
Residual variance*	255	85·181	·334		

* From Table II.

(*iii*) EFFECT OF THE INTERVIEW ITSELF

As was reported above, the questionnaire was given before each interview on 8 occasions and after each interview on the remaining 8 occasions. A balanced order of "before" and "after" interviews was operated. The total scores of the 8 "after" questionnaires were only slightly lower than the total scores of the 8 "before" questionnaires. The difference was 1·42. A difference of 3·49 would have been necessary to reach the 5 per cent. level.

These results are consistent with the possibility that there was on the whole, no immediate reaction to the interview, and that the discussions have their effects in the periods of no discussions.

(*iv*) EFFECT OF INTERVIEWER

Here, the order of placing the interviewer was confounded so that there is an overall bias in favour of the junior author. Analysis of any differences between interviewers is not possible however since the "before" and "after" arrangement of the questionnaires precludes isolating any one interviewer's effect. Such a measure could only be obtained if questionnaires were given before the first and after the second of the interviews consistently throughout the experiment. It might be argued that administration of the questionnaires by different interviewers might give rise to difference in scores. Analysis of the differences between the means of questionnaires given by the respective interviewers shows that these means differ only by ·028 whereas a difference of 3·49 would be necessary for the 5 per cent. level of significance, on a "t" test of the difference between total scores.

FURTHER OBSERVATIONS

A period of digestion and discussion of results ensued. Because of lack of time and the difficulty of some of the problems which arose, a few weeks were spent in this way. Exactly two weeks after the end of the experiment the patient was given the questionnaire again. Consultation of Table II and the Graph shows a further fall in the patient's strength of persecutory beliefs and still no change in his guilt.

Approximately seven weeks after the end of the first experiment another experiment was begun. It was intended to investigate the relative influence of the pause and the week-end. By this time the patient's co-operative attitude had evaporated and, after two discussions he pressed for their termination.

It was decided, on clinical grounds, to comply with this request. There are obvious lessons to be drawn from this failure. The results of the two questionnaire measures taken during this period are given in Table II and the Graph. The two interviews were directed at the discussion of guilt.

Clinical Outcome

It was not possible to carry out measures of overall behavioural changes during the course of the experiment. All that can be said is that, about two months after the end of the experiment, an improvement in the patient's mood and behaviour had been noted by Dr. Mallett. Dr. Mallett reports that the persecutory delusions were held less vehemently and less firmly than before. He was finally discharged three months after the last interview with the psychologists, as "much improved".

At the end of the experiment arrangements were made for the patient to work in the hospital gardens, and this he appeared to do very energetically. Arrangements were also finalized for the patient to go to an Industrial Rehabilitation Unit to train as a brick-layer. Dr. Mallett reported that the patient received these suggestions with "quiet enthusiasm".

Dr. Mallett had, of course, been seeing the patient twice a week regularly throughout his stay in the hospital. There is no way of determining the relationship of all these activities and of the experiment to the patient's clinical improvement. All one can say is that the discussions did not do him immediate obvious harm, and might have done him some good.

The patient was asked by Dr. Mallett, just after the cessation of the second experiment, whether he felt that the psychologist had been able to help him in any way. His reply was, "Yes, well, it did help to get rid of a lot of superfluous hate. I don't know where it came from, but it seemed to be there and it's better out". Towards the end of his stay in hospital he indicated that his delusions of persecution did not bother him greatly, and he said, "People have been working so hard to help me, I just can't believe there is any plot against me." These responses underline the necessity for developing measures of the patient's view of the experimenters and the total situation.

Qualitative Changes in the Paranoid Questionnaire Items

In view of the uncertainty surrounding the reactions to the depression and hostility items, and the lack of definite change on the guilt items, it is only worth while reporting on the qualitative changes in the paranoid items. The first time he was tested the patient sorted eight of the items under "definitely the case" (Category 4), five of the items under "may be the case" (Category 3) and five of the items under "not sure" (Category 2). The first group consisted of Nos. 2, 5, 6, 9, 14, 15, 16 and 17. These, with one exception, refer to the aims and activities of the persecutors. The third group, the "not sures", consisted of items 8, 12, 13, 14 and 18. These refer mainly to biological beliefs such as his own and his children's illegitimacy and his physical condition (see Table I).

On the sixteenth occasion of testing the patient sorted no items under Category 4, sorted items 7, 14, 17 and 19 under Category 2, and items 12, 13 and 20 under Category 0. This last category was sorted under the heading "definitely not the case". The Category 2 items refer to "biological beliefs". Of the Category 0 items, 12 and 13 refer to the wife's unfaithfulness and the children's illegitimacy. No. 20 refers to dope being put in the patient's food.

In terms of the questionnaire responses, the patient lost all tendencies towards the unqualified acceptance of his persecutory delusions which he previously possessed, and ended up by rejecting 3 beliefs, of which 2 had, from the beginning, been sorted under "not sure".

Discussion and Conclusions

First of all, it seems unlikely that the patient's questionnaire responses were deliberately contrived by the patient. The motive for such contrivance would presumably be to obtain a discharge from the hospital. For this purpose it would be necessary for the patient to pretend that the discussions were modifying his beliefs in the desired direction. If this were the case it would be difficult to explain why his persecutory beliefs became stronger after the discussion of guilt. It would also be difficult to explain why guilt was so little affected by direct discussion. The fact that the senior author felt himself relatively ineffective, at times, in the discussion of guilt is not a sufficient explanation. The patient would, if he were merely trying to satisfy us, have made some concessions. In fact, if out-argued, he often came back on the next session with a new and improved argument.

Secondly, it is possible that the overall downward trend might have occurred because the patient was becoming better for some reason completely unrelated to the discussions, or that he was merely reacting to the kind of social contact which the discussion provided. The fluctuations around the regression line might have been due to a peculiar concomitance of factors which might never again come together, and which had no relation at all to the content of the discussions. In fact the implications of our result could not be accepted without further experiment.

The results from an experiment on a single subject can only be accepted with confidence if they give rise to generalizations which enable one to make new and valid predictions about the behaviour of that subject. Such predictions must be about situations which the subject has not yet experienced. Unfortunately the patient's eventual attitude prevented us from doing this.

Thirdly, it is worth mentioning some of the ideas which do arise if we take at face value the explanation which provides the best fit for our results. One implication is that a series of discussions has to be followed by a period of no discussions if the full impact of the discussions is to be felt. An alternative implication is that the week-end is, for a complex of reasons, the period of psychological development, and that this effect is quite independent of whether or not discussions are taking place during the week-end. Our aborted experiment had been in fact devised to indicate which of these implications was the correct one. Another implication is that the activation of guilt feelings can result in the increase in the strength of paranoid delusion.

It is further appropriate now to recall that our original aim was to investigate the idea that the patient's paranoid beliefs would be abnormally resistant to the normal processes of modification. The results of the present experiment, if taken at their face value, do not give a clear-cut answer to this question. Only eight of the interviews were directed at the paranoid beliefs themselves. One is forced to wonder whether passionately held beliefs of any kind would have shown as much change in eight interviews as our patient appeared to show. Their abnormality, therefore, might lie more in their content rather than in their resistance to modification. This matter cannot be settled without further investigation and appropriate controls.

Fifthly, we should mention the fact that the patient, under the impact of the discussions, offered additional information of interest. The most unexpected item was his statement that his persecutory feelings were most intense after breakfast. He said he was extremely irritable at this time and this was the time when people seemed to be trying their hardest to get at him. A second admission of interest was that he felt much better after hard exercise in the open air. If we had had more time at our disposal we would have carried out an experimental investigation of these two assertions of the patient. We were not in principle confined to the investigation of the effects of rational argument upon persecutory beliefs: we were only committed to the bringing of these beliefs under experimental control, and in the pursuit of such a purpose we would always be thoroughly opportunist.

Finally, it should be pointed out that the findings reported in this paper do not warrant any conclusions about the nature of paranoid delusions. At best they can only be regarded as a starting point for further research. Our first objective is to put forward a point of view on research method. The experiment we carried out, with all its weaknesses, serves to illustrate the point of view which is being advanced. In addition the results warrant the conclusion that the continued development of the direct experimental investigation of psychological disorders might produce useful results. It would seem to be worth our while, therefore, to carry out further experiments on paranoid delusions in which more attention were paid to the details of planning and of quantifying all the relevant variables.

SUMMARY

This paper advances the view that the experimental clinical psychologist should concern himself, more than he has done hitherto, with the direct experimental manipulations of the psychological disorders which bring patients into the mental hospitals. As an illustration of this point of view an account is given of a preliminary experiment, with a patient diagnosed as a paranoid schizophrenic, on the effect of rational discussions upon persecutory delusions and feelings of guilt. The outcome of the experiment was consistent with the following conclusions:

(*i*) The rational discussion of paranoid beliefs diminished their strength.

(*ii*) The discussion of guilt had little effect on the strength of guilt feelings but increased the strength of persecutory beliefs.

(*iii*) The effects of a series of discussions were delayed and appeared only after a period in which there was no discussion.

It is made clear that further experimentation is necessary before the generalizations described above could be accepted with any confidence, and that the results serve, primarily, to indicate that the direct experimental analysis of psychological disorder might lead to useful results.

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STUDIES IN SCHIZOPHRENIA, V.

DISTURBANCES OF PROTEIN AND TRYPTOPHAN METABOLISM AND TOXICITY OF URINE*

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THE problems of diagnosis, pathogenesis, aetiology and successful treatment of the idiopathic schizophrenias is one of the most urgent and formidable ones which psychiatry has still to tackle. It is most urgent because of the large number of sufferers struck by the disease, made unfit for a successful life at an early age, and the distress imposed on them and their relatives. It is most formidable because our essential knowledge of the nature of this disease, or possibly group of diseases, is scanty and disorderly and opinions are conflicting and contradictory. This unsatisfactory state is made worse by the fact that there is quite an amount of disagreement on diagnosis, so that often the findings of different workers do not refer to comparable groups of patients (and conditions in which they are examined) which must add to the confusion, particularly when metabolic examinations are carried out. Too often "marginal psychoses", and also not rarely, paranoid hallucinatory states with and without changes of the personality are called "schizophrenia". The last-named states are not infrequently schizophrenoid reactions (or reactions of a schizoid person) to psychogenic causes or to potentially diagnosable physical disorders. On close clinical examination one would also find that the symptomatology of such cases is not that of idiopathic schizophrenia. In the "classical" schizophrenias of Kraepelin, Bleuler and Kleist, despite some discrepancies, the typical parafunctions of feeling, willing, thinking and activity, usually accompanied by hallucinations, mainly auditory, and paranoid delusions are present and also a disturbance of the albumin/globulin ratio in C.S.F. and serum. In the absence of this mental and physical symptomatology the diagnosis of idiopathic schizophrenia should not be made (Fleischhacker *et al.* (7, 8)).

The main factors alleged to produce idiopathic schizophrenia (usually in mentally and physically predisposed persons) are:

1. Psychogenic.
2. Encephalic-constitutional.
3. Extra-cerebral (vascular, metabolic, infectious, toxic).

1. "Psychogenic" disorders do not show symptoms of an "organic" character as seen in the schizophrenias, such as the typical para-disturbance of speech and thought, a number of motor signs, reflex abnormalities, slight

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* Based on a paper read by H. H. Fleischhacker at the Second International Congress for Psychiatry, Zurich, 1–6 September, 1957, and dedicated to Prof. K. Kleist on his eightieth birthday.

atrophies of the brain and some metabolic dysfunctions. A mainly psychogenic cause is therefore unlikely, unless one believes with C. G. Jung (20) that psychogenic processes in certain persons can produce a toxic process which will then cause an organic disease of the brain.

2. The idea of a constitutional degenerative cerebro-systemic disease, *per se*, is based on the findings of hereditary research, of body-build and on the just mentioned "organic" signs found in the idiopathic schizophrenias; but does the course of the illness justify the claim? If at all, only for those cases which begin insidiously and progress more or less chronically though not necessarily continuously.

3. But there are the many cases with a more violent start, which, at least for practical purposes, are followed by a full remission with, or often without any real treatment. There may be more relapses and remissions before the state becomes "chronic", and even then late remissions may occur. Such a course is scarcely ever seen in constitutional or hereditary primary atrophic diseases of the C.N.S. such as muscular atrophies, lateral sclerosis, Huntington's chorea or Friedreich's ataxia. Such a course occurs, however, in non-constitutional diseases of the C.N.S. such as disseminated sclerosis, arteriosclerosis, hypertension and a small number of G.P.I.s. In general illness it occurs in some chronic infections like tuberculosis, or "allergic" disorders, rheumatoid arthritis, or in metabolic ones, like gout, when "external" pathogenetic factors act as either the cause, or at least as the precipitant of the illness. It appears, therefore, justified to assume that in idiopathic schizophrenia some exogenous, or at least extracerebral factors (in the widest sense of the term) play a role, and it is with this pathogenetic aspect of idiopathic schizophrenia that this paper is concerned.

In active idiopathic schizophrenics an increase of globulins is always found in the serum (Fleischhacker (9) and the C.S.F. with a slight increase of round cells in the C.S.F. (Fleischhacker (7)). With improvement of the clinical state the absolute and relative increase of globulin diminishes and may disappear from the serum, whilst in the C.S.F. the increase usually still can be found for a longer period, though to a lesser extent. Although this disturbance of the A/G ratio in these patients is characteristic only and non-specific, the regularity of its presence makes one feel that its elucidation might lead to a better understanding of the pathogenesis of the disease (or group of diseases).

Like some others, one of us (H. H. Fleischhacker) once thought that the cause of the disturbed A/G ratio might be sought in a vascular disturbance of liver function. The hypothesis was tested some years ago (9) on about two dozen idiopathic schizophrenics and a larger number of controls which included mentally normals with slight myxoedema and chilblains, and organic psychotics with and without hypertension, with and without heart-failure, with and without enlargement of the liver. But no regular correlation between peripheral and/or central-vascular disturbance and increase in globulins was observed. Meanwhile, it had become clear that the increase of globulins was essentially of the γ fraction, which raised the question of a possible infectious pathogenesis, and it was decided to check the claims of some workers in this respect.

In order to test Pickworth's (25) claims of focal infections, a number of schizophrenics and organic controls had their sinuses and teeth X-rayed; no regular correlation between increase of globulins and infection of these parts could be established (Fleischhacker (9)).

Papez's (24) claims of a so-called zooid infection of the brain could not be confirmed either: the phase-contrast microscope showed the presence, though less in number, of bodies similar to those described by Papez in the brains of schizophrenics, in emulsions of healthy guinea-pig brains which had been removed aseptically immediately after the animals had been killed (Fleischhacker (10)).

Dr. Cathie, of the Hospital for Sick Children, London, kindly carried out tests for toxoplasmosis on 12 selected schizophrenics: the number of positive tests corresponded to that of the population in general.

With Dr. Gurassa one of us (15) checked Rosenow's claim of special α-haemolytic streptococcal infection in schizophrenics and epileptics; the tests showed no specific significance but, interestingly, the number of positive results was higher in schizophrenics and epileptics than in the controls (organic psychoses, neurotics and normals).

That these few investigations have so far led to negative or contradictory results, does not necessarily preclude further investigations. One may not have searched for the right agents, or, again, the infection need not be "specific". It is clear, however, from the neuropathological aspect that, if there is an infection at all, it cannot affect the brain directly, but only indirectly by originating a toxic process, as in many of the acute infectious-toxic psychoses, possibly via the intestines. Now, it has been noticed (Fleischhacker (9)), first by chance, later on in more systematic investigations, that the faeces of newly admitted organic psychoses, schizophrenics and epileptics contained non-lactose fermenting bacteria more often than those of patients afflicted with neurotic and cyclic disorders. Although this variation in intestinal flora may be caused by the disease *per se*, by influencing intestinal functions, metabolism and flora it recalls the claims of Reiter (26), Buscaino (4a), de Jong (19) and others that infectious and other disturbances of the intestines may give rise to a faulty metabolism with a toxic action on the brain. Berger (2), Weichbrodt (36), Gamper and Kral (12), Baruk (1a), and more recently other Continental (Rieder (27, 28)) and American workers have published work on the toxicity of body-fluids from schizophrenic and other psychoses on plants, animals and even humans (Heath *et al.* (16)). For more references see Fabing (6). Connected with the problem of "toxicity" is the problem which substance or substances may act in a "toxic" way. In this respect the question of a disturbed tryptophan metabolism has been re-raised recently by several workers (4a). So before going any further it appeared desirable to examine the problem of "toxicity" and of toxic substances.

Hoffer and his colleagues (18, 23) had pointed out that some of the fashionable hallucinogens and adrenochrome contain an indole-ring. It is felt strongly by at least one of us (H.H.F.) that the so-called model-psychoses with the prominence of disturbances in the optic field are more related to the exogenous reaction type of Bonhoeffer than to idiopathic schizophrenia. Other authors like M. Bleuler and I. Sano have expressed similar opinions. Riegelhaupt (29) in this laboratory uninfluenced by clinical considerations, however, applied the glyoxylic acid test for tryptophan and some of its metabolites on the urines of a number of patients and normals. He found the test positive, indicating most likely a disturbance of tryptophan metabolism, in most of the unselected schizophrenics, a few organic psychotics, and negative in physically healthy normals, neurotic and cyclic patients. Corresponding results have been described by the McGeers (22) and other workers, using paper-chromatography techniques. In detail the results of these workers are still conflicting (Sano (31), Buscaino (4), Riegelhaupt (30)) and others (see Fabing (6)).

In view of these findings from this and other laboratories it appears justified to formulate the whole problem in the following way:

Has the increase of γ globulins in the sera and C.S.F.s of active idiopathic schizophrenics the significance of an immune body, not necessarily produced by one specific infection, but by several different ones, and amongst them by a changed intestinal flora? This could disturb tryptophan metabolism and then produce abnormal metabolites with a direct toxic influence on the brain (and perhaps other organs too); although there is the possibility that it may interfere with vitamin B metabolism and thus lead to a disorder of brain function. In this paper only part of the problem has been attempted*:

1. Are urines of schizophrenics "toxic"?
2. Is this toxicity connected with a positive glyoxylic acid reaction?
3. Is there any connection between these two factors and the raised γ globulin-level in the serum?

It was decided to carry out experiments on patients suffering from different diseases, and on schizophrenics at different stages of their illness. Consequently, the following examinations were carried out on 70 persons, namely: senile dementias (9), Huntington's chorea (7), paralysis agitans (2), hypertensive psychoses (9), epileptics (13), schizophrenics (17), some other patients (7), and six members of the laboratory staff. Beyond this group the g.a. reaction was applied to the urines of about another 100 persons, including other members of the hospital staff (nurses), neurotics and cyclic patients and other schizophrenics. These are not listed here, but reference will be made to the results.

The examinations carried out were:

1. *Urines*

(*a*) Chemical: indican and glyoxylic acid (g.a.) reaction.

(*b*) "Toxic": influence of urines on the growth of yeast, following Rieder who had measured the respiration of yeast (27). We are, of course, well aware of the fact that any effects urines may, or may not, have on the growth of yeast are not necessarily applicable to the human brain.

2. *Serum*

Disturbance of the A/G ratio as shown by Thymol and Modified Takata-Ucko-tests.

TECHNIQUES

1. "Glyoxylic acid" reaction on the urine: Modification of Riegelhaupt's (29) technique:

Reagents:

1. Sulphuric acid conc.
2. Copper sulphate solution, 2·6 per cent.
3. Glyoxylic acid (g.a.) reagent (B.D.H.), or a suitable brand of glacial-acetic acid; or water.

To 2 c.c. of urine adjusted to specific gravity 1·010 or as near as possible are added 1 ml. of g.a. reagent (or 1 ml. of glacial-acetic acid, or 1 ml. of water) and 0·1 ml. of a 2·6 per cent. solution of Cu SO_4 and well mixed. The test tube is then submerged in a sloping position into cold water and 1 ml. of conc.

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* Examinations on the possible connection between intestinal flora, A/G quotient in serum and some aspects of tryptophan metabolism are in progress.

H_2SO_4 is allowed to run in within 30 or 40 seconds. One minute after the addition of the H_2SO_4 the reagent mixture is thoroughly stirred with a glass rod, the test tube removed from the water and put into a rack and the colour read after 15–20 minutes. Hereafter 2 ml. of chloroform or carbon-tetrachloride are added to the test tube and the mixture is vigorously shaken for 15–20 seconds and then centrifugalized.

The colours of the supernatant, and of the chloroform layer are noted and compared, and this with that of the *Obermeyer reaction:* To 2 ml. of Obermeyer reagent are added 2 ml. of urine of the same specific gravity as under (1). The reaction at the interface is observed for 5 minutes, then 2 ml. of chloroform are added and the mixture is shaken and centrifugalized.

Some of the modern tranquillizers may render false positive in both tests.

Evaluation: of the g.a. reaction: essentially brown: negative; essentially red-brown: doubtful; mauve or pink with a slight admixture of brown: weak positive; essentially mauve: positive.

2. *Modified Takata-Ucko Test* (3)

Reagents: Sodium carbonate: 0·36 g. of the anhydrous salt (Analar) is dissolved in distilled water to 100 ml.

Mercuric chloride: 0·50 g. of the salt (Analar) is dissolved in distilled water to 100 ml.

These solutions should be stored in the refrigerator and renewed when precipitation or discolouration occurs.

Method: A 1/4 dilution of fasting serum is prepared by adding 0·25 ml. serum to 0·75 ml. physiological saline in a Wassermann tube. After mixing, 0·5 ml. is transferred to a second tube containing 0·5 ml. saline. Four further twofold dilutions are prepared, giving altogether dilutions from 1/4 to 1/128. 0·1 ml. sodium carbonate solution is added to each tube and thoroughly but gently mixed, followed by the addition of 0·1 ml. mercuric chloride solution and mixed similarly. All tubes are allowed to stand at room temperature and observed for precipitation at 30, 60 and 90 minutes and at 18 hours. Precipitation is seen first as a sharply demarcated clear zone at the "corners" of the meniscus, which gradually extends down the tube.

Evaluation: a test tube is counted as sedimenting when there is a clear separation.

Test *Negative:* not more than 2 sedimentations after 90 minutes, not more than 4 after 18 hours.

Test *Positive:* at least 3 sedimentations within 90 minutes.

Test *Doubtful:* not more than 2 sedimentations within 90 minutes, not more than 5 after 18 hours.

Test *w. Positive:* not more than 2 sedimentations within 90 minutes and 6 after 18 hours.

3. *Thymol Turbidity Test*

Reagent: Add 500 ml. distilled water to 1·38 g. barbitone and 1·03 g. sodium barbitone and approximately 3 g. thymol. Bring just to boil, shake well and then cool thoroughly. Seed with a small amount of powdered thymol and stand overnight at 20–25° C. Filter after thorough shaking.

Method: Add 6 ml. buffer to 0·1 ml. serum. Mix well and stand for 30 minutes. Compare visually or photoelectrically (red filter) with protein solutions containing 10, 20 . . . 100 mg./100 ml. as used by Kingsbury. Thymol turbidity units=protein equivalent/10. If necessary dilute with more buffer and apply the appropriate correction to the final reading.

Evaluation: Negative: 0–2 units; doubtful: 3 units; positive: 4 and more units.

4. *Yeast*

(a) *Preparation of Standard Suspension. Candida pseudo-tropicalis* was selected as the test organism, because it is less likely to be affected by conditions outside experimental control. The stock culture on oxoid malt agar was stored in the refrigerator and sub-cultured on the day preceding the test on to the same medium and incubated at 37° overnight. On the morning of the test the organism was emulsified in sterile physiological saline to a density equivalent to the No. 3 Browns Opacity tube.

(b) *Preparation of Urines.* First morning specimens of urine were adjusted to pH 5·0 with hydrochloric acid and to SG 1·010 with distilled water. After Seitz filtration (Ford Sterimats G.S.) 10 ml. amounts were distributed under aseptic conditions to each of three universal containers.

Growth of Yeasts in Urines

Two of these three 10 ml. amounts of urine were inoculated with 0·1 ml. standard yeast suspension under aseptic conditions. The third 10 ml. samples provided "blanks" for subsequent turbidimetry.

All tubes were incubated at 37° in a water bath with occasional agitation.

(c) *Turbidimetry.* One ml. of well-mixed test yeast culture was removed under aseptic conditions from each of the two tubes. Optical densities were determined, using 1 ml. fluid from the third tube as "blank". Densities were determined at first after 0, 3 and 20 hours incubation at 37°. All readings were taken on the Spekker Absorbtiometer using micro-cells of optical length 2 cm. and filters Wratten No. 2 and Chance No. 3. It was found that there was generally good agreement between the duplicate tubes at 20 hours but that results after 3 hours incubation were not always satisfactory, and for this reason the 3 hour readings were later discontinued.

Results

Before the results are given in tabulated form a few general remarks in connection with the introduction may be allowed.

It is important to know the mental, physical and therapeutic state of the patient. Some of these factors may have a considerable influence on the outcome of all the metabolic tests used in this (and similar) examinations: e.g. Largactil and similar drugs may interfere with the colours of the g.a. reaction; they also slightly influence the A/G ratio. Also some physical disorders produce positive tests in urine and serum. The growth of yeast in urine is influenced by its pH, specific gravity, sugar content, vitamins, amphelamine, digitalis, etc. but not by barbiturates.

On the whole, g.a. negative urines produce a "normal" growth of yeast, while—compared with this—the growth of yeast is depressed in g.a. positive ones. But in a number of conditions such as mentioned above and possibly many others too, discrepancies will occur and g.a. positive urines may produce a normal, or even "supranormal" growth of yeast. On very rare occasions we have seen the opposite: depressed growth of yeast with g.a. negative urines.

If, therefore, the group of patients examined is comparatively small and consists of persons suffering from the same disorder but in various mental, physical and therapeutic states the individual result and average of the "group" may be considerably influenced, and "statistical analysis of small numbers" may be misleading as can be seen from the following two examples:

TABLE I

1. Evaluation of reactions:
Growth of yeast: + or − per cent. normal.
Others: normal=0 or 1, upper border line=2.
Weak positive=3, positive=4.

			g.a.	Yeast (Per cent.)
2.	(*a*)	5 Seniles: no sugar in urine	3·8	−18
	(*b*)	4 Seniles: with diabetes	2·6	+80
	(*c*)	Average of whole group	3·3	+25
3.	(*a*)	8 Subchronic schizophrenics	3·8	−15
	(*b*)	9 Schizophrenics after treatment, etc.	2·5	− 2
	(*c*)	Average of whole group	3·1	− 8

1. *Seniles:* 4 out of the 9 patients were diabetics; yeast grew freely and consequently the average of the whole group with regard to yeast gives a wrong impression, in comparison with the positive g.a. reaction.

2. *Schizophrenics:* There were 17 patients. Eight of these were active "subchronics"; of the other nine 2 were very acute: 5 were after treatment with clinically little change, 1 was a "burnt out" chronic patient, another had practically recovered; in these last 9 patients the g.a. reactions were nearly normal and again a biased picture is conveyed for the whole "group". The Takata reactions with the exception of the "burnt out" and the "recovered" patient were pathological in all the schizophrenics.

It may be mentioned here that 3 schizophrenoid psychoses were practically negative with all reactions.

Now to the results of the main groups:

TABLE II

	Urine			Serum	
	Indican	g.a.	Yeast (Per cent.)	Thymol	Takata
9 Seniles+2 paralysis agitans	1·0	3·4	+20	1·3	1·6
7 Huntingtons	2·0	3·8	−20	2·3	1·6
9 Hypertensives ..	2·0	3·9	−35	2·5	1·9
17 Idiop. schizophrenics	1·5	3·1	− 8	2·1	*3·3*
3 Sympt. schizophrenics	2·0	2·0	− 6	1·3	1·4
13 Epileptics	0·7	3·3	−14	1·2	*3·2*
6 Healthy normals ..	2·5	1·5	normal	normal	normal
4 of these with colds, etc.	2·0	3·5	−15	not done	not done
4 Miscellaneous (organic)	1–4	2–5	−30 to +400	1–2	0–2

RESULTS

(a) *Indican*

It is clear, and from individual examinations still more than from groups, unless it is present in large amounts, that there is no connection between this substance and the outcome of the g.a. reaction.

(b) *"G.A." Reaction*

The reaction was normal in healthy normal people, and we have confirmed in many cases not listed here, that it is also negative in neurotics, even schizoid neurotics, when they are physically healthy; the same holds good of uncomplicated cyclothymic disorders. The reaction can, however, become positive in normal people, who are somatically ill, e.g. when suffering from a severe cold, influenza or some form of cystitis, and possibly hypertension. A previously negative g.a. reaction may also become positive under "modified" E.C.T. In active schizophrenics the reaction is positive as a rule, exceptions to be discussed later, and also in a number of epileptics and "organic" psychoses of primary (senile dementia, paralysis agitans, Huntington's chorea) or secondary neuronic disease (hypertensive encephalopathy, possibly also post-encephalitis and chorea minor).

(c) *Toxicity of Urine and "G.A." Reaction*

As already stated, the growth of yeast is usually "normal" in g.a. negative urines, but becomes depressed in g.a. positive ones. Thus, growth is normal in the urines of healthy, normal persons and physically healthy neurotics and uncomplicated cyclothymics; it is depressed in the urines of idiopathic schizophrenics, epileptics and other organic psychoses, and also in normals, suffering severe colds, influenza and from some types of cystitis. Alcohol intake may provide pathological urines. A number of causes when discrepancies occur have been mentioned above. It should be added that tryptamine and an, even very slight, increase of the concentration of copper depresses the growth of yeast (whether this is a possible explanation of g.a. negative urines with diminished growth of yeast, we cannot say for the time being). Whether in g.a. positive urines the "toxic" reaction on yeast is therefore due to the substances giving a positive glyoxylic acid reaction alone, or in combination with other substances, e.g. copper, or whether, with regard to toxicity, a positive g.a. reaction acts only as an "indicator" of other "toxic" metabolites is still unknown.

Our findings of a "toxicity" of urine together with a positive g.a. reaction in schizophrenics confirms the corresponding claims of other workers (1, 2, 12, 16, 27, 36). But it is clear from our controls that neither "toxicity" of urine nor a positive reaction is specific of schizophrenia. There are parallels to this in the findings of previous workers. Gamper and Kral (12) using C.S.F. on young mice found this toxic in schizophrenics but also in other organic C.S.F.s. Baruk and Camus (1a) working on the toxic and catatonic effect of bile have shown that such effects occur not only with the bile of schizophrenics but also with that of other patients.

Our results are possibly particularly closely connected (paralleled) with those of Heilmeyer and his school (17) on copper metabolism; they found the serum level of copper increased in many organic states and also in schizophrenics, and (in contrast to our findings) in cyclic disorders. They may also be similarly connected with the findings of Leach *et al.* (21) on ceruloplasmin, adrenochrome and glutathione levels in schizophrenics and in normals suffering from organic disorders. The authors assume that a general disturbance of oxidation may be at play. The fact that copper in concentrations of about twice the normal level in urine depresses the growth of yeast may be recalled here.

(d) *The Turbidity Reactions*

Both reactions (Takata and thymol) were usually normal in the senile group. In hypertensives and Huntington's chorea the Takata was either normal or borderline, but the thymol was often at the upper limit of the norm or weakly positive, and one wonders whether the weakly positive thymol (which test is slightly less sensitive towards γ globulin than the Takata modification used in this laboratory and possibly more sensitive to an increase of β and γ globulins and lipoids) has any connection with the arteriolar changes found in both diseases, in their action on kidney and liver. In active schizophrenics and epileptics, however, the Takata reactions are as a rule positive, with the thymol tests being more often doubtful or normal respectively, and this increase in γ globulins differentiates these patients quite clearly from all the other psychoses (organic or functional) examined in this series.

CONNECTION OF RESULTS OF SERUM AND URINARY REACTIONS

While toxicity of urine towards yeast and positive glyoxylic acid reaction run as a rule parallel, a similarly close parallel does not exist when urinary and serum reactions are compared. As even thymol and Modified Takata-Ucko-test (MTUT) results are not always in agreement, and as a positive MTUT is more sensitive to an increase of γ globulin and more characteristic of idiopathic schizophrenia than a positive thymol reaction, we shall neglect in the following discussion the results of the thymol reaction; though in organic vascular disease this may be of higher importance.

In principle: positive urines (glyoxylic acid and "toxicity") may occur with quite negative or only doubtful MTUTs as was seen particularly in Huntington's chorea and senile dementia. Positive MTUT and negative urines scarcely ever occurred in the organic group.

On the other hand, in the 8 uncomplicated active schizophrenics of this series positive MTUT occurred together with positive urines. When the illness improves all reactions may become negative. The same occurs possibly, when the illness ceases to be active and the patient appears to be in an arrested state of chronicity. This confirms previous unpublished findings. However, after insulin and largactil treatment without noticeable change of clinical behaviour (though there may be some on very close examination) and possibly in rather acute states of illness, discrepancies were seen, in that the MTUT was positive while the urines were negative. As this has happened altogether in 7 cases so far only it may be rash to comment upon this discrepancy, were it not for the following reasons:

1. Some of the "tranquillizers" e.g. reserpine and largactil, are said to influence serotonin metabolism (31); (may not the same hold good for insulin shock treatment?).

2. It has been claimed (although so far not confirmed) by the late Dr. Sherwood (32) that in stress the excretion of 5HIAA is low, while it is usually high in schizophrenics and carcinoid tumours. Our 2 very acute patients were apparently very "stressed".

On the ground of the present and former experiences in this laboratory with the MTUT and the glyoxylic acid reaction positive in schizophrenics it appears therefore that as a rule positive urines and MTUT in these patients occur together, but that in certain conditions discrepancies will arise.

The occasional independence of the g.a. reaction of an increase of γ globulin in schizophrenics is remarkable for another reason: in that γ globulins have a very much higher concentration of tryptophan than other protein fractions; so it does not appear as if the increase in serum of this globulin is a direct source of the substances that gave a positive g.a reaction.

Altogether, it is therefore clear, as was already evident from some of the organic psychoses, that an increase of γ globulins, on the one side and on the other side positive glyoxylic acid reactions in the urine and depression of growth of yeast by such a urine, are not necessarily interdependent and that at least two metabolic systems are involved which are usually but not always both disturbed in active schizophrenics.

ORIGIN AND SIGNIFICANCE OF THE REACTIONS

(a) *G.A. Reaction and Toxicity*

How and where the disturbance producing positive g.a. reactions occurs, is for the time being quite obscure, with the great variations of diseases in which the disturbance has been observed. Infections in normals, or quite differently, primary (Huntington, senile, paralysis agitans) and secondary (hypertension, chorea minor, post-encephalitic states) neuronic disorders may all produce g.a. positive and yeast-growth depressing urines besides organic diseases of unknown origin like idiopathic schizophrenia and epilepsy (whether with regard to epilepsy, idiopathic or traumatic there is a difference we cannot say yet). It may be assumed that "positive" urines may be obtained in many other conditions.

Assuming that the substances depressing the growth of yeast are toxic to brain metabolism and that they are tryptophan metabolites, the following about the places of their origin may be stated:

Tryptophan-splitting enzymes have been found present in the liver, the kidneys and intestines, and a number of intestinal bacteria. One has to expect some renal, hepatic and intestinal (atrophy) damage in the senile group. Kidneys and liver are practically always affected in hypertensive and arteriosclerotic patients and also possibly in a number of even younger patients with Huntington's chorea, in whom arteriofibrosis when present is often not only restricted to the brain (Fleischhacker, unpublished). We have also found for many years that the urines of quite a number of our younger chronic schizophrenic patients contain from time to time, without any obvious reason, traces of protein (which however do not directly cause a positive g.a. reaction), scanty red cells and hyaline casts. It should also not be forgotten that mentally normals afflicted with some infection and consequent metabolic disorders produce "g.a." positive urines, which for the time being cannot be differentiated from that of psychotic patients.

On the other hand, all the patients with organic mental disease tested in this examination have pathological processes in their brains, which affect basal ganglia and diencephalic structures with their ascending and descending connections, and often also some cortical (orbital, cingular) areas which are all potentially regulating or at least influencing metabolic processes. So, while tryptophan metabolism takes place in the periphery there is the possibility that this is influenced by central nervous processes. The problem looks very complicated indeed.

What is the probability that the substances giving a glyoxylic acid positive and "toxic" urine produce psychotic symptoms? For the time being our experience is not large enough to answer the question definitely.

However, most of our g.a. positive epileptics were in the hospital rather for behavioural difficulties than for frank psychotic symptoms. Also some of the organic psychoses were not hallucinated. Furthermore, so far, we have failed to obtain a convincing positive glyoxylic acid reaction (unless produced by proteins) in sera diluted from $\frac{1}{2}$ to 1/200, or from a few C.S.F.s from urine-positive patients. Although more experience (including technical problems) is necessary, some caution appears desirable with regard to the question which is the primary process producing the urinary abnormalities described in this paper and on the significance of these findings with regard to the production of psychotic symptoms.

(b) *The Turbidity Reactions*

These reactions, particularly the MTUT when positive, are indicative of an increase of γ globulins. Their organic and pathogenic significance in idiopathic schizophrenics (and a number of epileptics) is just as obscure as that of the positive g.a. reaction and disturbance of tryptophan metabolism discovered by paper chromatographic techniques. From what we have found, it does not appear as if the disorder producing g.a. positive and toxic urines, produces also the increase of γ globulins in the serum and C.S.F. by necessity. Still, it is worth while to discuss some of the possible sources of the disturbed A-G quotient.

1. *Adreno-cortical dysfunction.* There is still some discrepancy as to whether and how far this influences the level of γ globulins. Also, the increase of this protein fraction is found regularly in idiopathic schizophrenics and occurs usually more frequently in them than adrenal dysfunction; vice versa the MTUT in sera and C.S.F. of physically healthy neurotics is negative, while adreno-cortical dysfunction has been described in such patients.
2. *Vitamin dysmetabolism.* Vitamin B_6 and nicotinic acid are derivatives of tryptophan. Vitamin B_6 is supposed to influence the A/G ratio. No such disturbances have so far been described in schizophrenics. Still, one should keep an open mind in this respect, as e.g. vitamin C stores in chronic but well-nourished schizophrenics are often somewhat diminished in Spring when compared with those of other, even "organic", patients from the same ward (H.H.F., unpublished). Disturbances of vitamin C in serum have also been described (21).
3. *Cerebral function.* Next, one may ask whether disturbance of cerebral function can influence globulin-metabolism. The influence of hypothalamic function on antibody formation is still under discussion (5, 33). But Vogt (34) and particularly v. Wahren (35) are quite definite that the hypothalamus is not affected in schizophrenia while quite a number of cortical and subcortical regions are; some of these (orbital areas, the cingular system, some mesencephalic and diencephalic nuclei) have an influence on metabolic processes. On the other hand, these regions show very often pathological changes in many organic psychoses such as examined here (Huntington's chorea, senile and hypertensive disorders), and yet in them disturbances of the A/G quotient were absent, or if present rarely of the order of those noticed in schizophrenia (and most of the epileptics tested in this investigation), while g.a. positive and "toxic" urines were frequently found. So, if there are central factors influencing protein and tryptophan metabolism at all, they are unlikely to have identical localization in the brain.

4. *Liver metabolism, R.E. system and infection.* It is reasonable to assume, particularly in connection with the work of Georgi and his school (13, 14) that the disturbance of the A/G ratio is connected with dysfunctions of the liver, which may also account for the disorder of tryptophan metabolism. But what is causing disturbance of liver function? According to the experiments described at the beginning of the paper, a vascular disturbance can be ruled out. The fact that an increase of γ globulins was present in most of the epileptics tested, may make one think that some liver-functions are deranged by chronic intake of barbiturates, and have nothing to do with the disease proper. But a few neurotics who had been on barbiturates for a very long time had quite normal Takata and thymol reactions and g.a. negative urines. Also many schizophrenics who had their sera and C.S.F.s examined had not been on barbiturates at that time, nor previously. Is possibly the disturbance caused by an "irritation" of the R.E. System not only of the liver but quite generally and including that of the brain (the cell increase in the C.S.F.), and is this possibly due to a chronic infection and/or intoxication producing antibodies? We do not wish to repeat the arguments in favour of this hypothesis as put forward in the introduction to this paper, but we think it permissible to add a few clinical observations:

1. It appears that quite a number of schizophrenics before the outbreak of their illness, though not necessarily immediately before, have undergone numerous infections and during the first years of their illness, when measured carefully, show occasional unexplained sub-febrile temperatures.

2. In the chronic state, however (and the same may hold good for epileptics) they are much less liable to succumb to slighter infections, particularly colds, influenzas, sore throats, etc.

3. Rosenow antigen-antibody skin tests, though by no means specific, were more often positive in schizophrenics (and epileptics) than in normals, neurotics, cyclothymics and a number of organic psychoses (Gurassa and Fleischhacker (15)). It appears therefore still worth while to pursue this subject, particularly as some infections produce positive glyoxylic acid reactions.

SUMMARY

1. The object of this paper was to find out whether there is any regular connection between an increase of γ globulin in the serum, positive glyoxylic acid reaction and toxicity of urine in idiopathic schizophrenics and other mental patients.

2. The following tests have been carried out in as near as possible similar experimental conditions on 17 idiopathic schizophrenics at different stages of illness and 53 controls, including 6 normals.

(*a*) The "glyoxylic acid (g.a.)" reaction on urines; a positive result is most likely indicative of a disturbance of tryptophan metabolism.

(*b*) The growth of yeast in urine; in order to find out whether and when there is any "toxic" (depressive) action of urines on the growth of this organism.

(*c*) The Modified Takata-Ucko Test (M.T.U.T.) and the thymol reaction on serum; positive reactions are indicative of an increase of γ, or β and γ globulins, respectively.

3. The g.a. reaction is as a rule, though with some exceptions, positive in idiopathic schizophrenics; it is also positive in epileptics, in other organic psychoses of primary and secondary neuronic origin and also in mentally normals when they are physically ill. The reaction is negative in physically healthy normals, neurotics and uncomplicated cyclothymics.

4. The growth of yeast is, as a rule, depressed in g.a. positive urines. There are, however, exceptions from this in a number of circumstances which are discussed.

5. How far the substances giving a positive g.a. reaction are solely responsible or co-responsible for, or only indicative of other substances producing "toxicity" towards yeast is not yet clear, nor whether these substances can produce psychotic and hallucinatory symptoms particularly of the auditory type.

6. The turbidity reactions in serum, particularly the M.T.U.T., are positive in active idiopathic schizophrenics (and also often in epileptics), very often together with g.a. positive

and "toxic" urines. But there are discrepancies between the outcome of serum and urinary reactions.

7. In most of the organic psychoses M.T.U.T. and thymol reactions were negative or only doubtful together with g.a. positive and "toxic" urines.

8. It occurs from (6) and (7) that serum and urinary reactions are not necessarily dependent on each other.

9. No definite conclusions can be drawn, for the time being, on the origin or the pathogenetic or aetiological significance of any of the positive reactions in the conditions examined. But there is no doubt that positive reactions, particularly a positive M.T.U.T., are diagnostically characteristic, though not specific, of uncomplicated active idiopathic schizophrenics.

10. It is remarkable that, so far, all the reactions performed have been positive only in people suffering from some kind of physical disease.

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THE INCIDENCE OF MENTAL DISEASE AMONG REFUGEES IN NORWAY*

By

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1. History

In Norway, the refugee problem has not been quantitatively significant. On the other hand, we have many examples to show that refugee work and interest for the fate of refugees has had an important position in the awareness and interest of the Norwegian people. The fact that the Nobel peace prize has twice been awarded to the High Commissioner for refugee organizations also speaks for itself. After the Russian revolution and its suppression in 1905 a small wave of emigrants came to Norway, and likewise after the 1917 revolution. Moreover we note a very limited and controlled immigration of victims of Nazi oppression from Germany and the occupied territories until World War II. After the war Norway was faced with a series of problems in connection with the refugees. In the first place it was a question of repatriation of Norwegian prisoners in Germany and refugees in Sweden, there were the forced evacuees from Finmark to be sent home, and then, last but not least, all the foreigners who had involuntarily landed in Norway during the war. Of these 140,000 were "displaced persons". These had been brought to the country by the Germans either as civil workers or forced labourers in "Organization Todt", "Organization Speer" or as prisoners of war, mainly Serbs (Jugoslavs), Russians and Poles. According to a list published in the annual report of the Ministry of Labour for 1947 (1948) the following figures are given:

Soviet subjects	about	84,000
Other prisoners of war	about	3,000
Civil Germans	about	10,000
Labourers in Organization Todt	about	31,000
Foreign forced labourers	about	13,000
Approximately		141,000

The living conditions of the majority of these prisoners is no doubt familiar to all, but it may be of interest to recall some of the circumstances in order to obtain a glimpse of the background from which the first group of refugees in Norway came. Kobro (1945) has described the conditions in a camp for Russian prisoners of war in southern Norway, and Kreyberg (1946) has given an account of the release of the allied prisoners of war in the north of Norway. From this last book we quote the concluding observations made by Frostad in his report: "The situation in the Russian prison camps was very bad everywhere with regard to living-conditions and clothing. Illness and death were widespread. The quantity of available medicine was insufficient in all camps, food and medical help is desperately needed."

The necessary help was given and in the course of a few months the majority of foreigners were either home again or on their way home, but a number of refugees had no desire to leave Norway. The reasons for this were many. For some the word "home" had become an empty term, or one that could only be associated with persecution and ruin, where the whole milieu in which one had lived and grown up no longer existed, where family, relations and friends were destroyed and where the only place one could hope to find a remembrance of one's loved ones was the cemetery or some names carved on a memorial.

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Others refused to leave because of the changed political situation in their home countries, this applied in the first place to Poles and partly to Jugoslavs, and others would not return home to the old political conditions after having become acquainted with the Norwegian people and its democratic government and way of thinking. Besides these rather "general" reasons, there were some rather more personal reasons which caused a number of young men to prefer to remain in Norway. There were connections made during or after the war. Some of these had a really romantic quality about them. Especially should be mentioned the many young girls who risked their liberty in order to give food to the prisoners. Some of them were also arrested and put into different camps in Norway where, by the most extraordinary means, they kept up the contact with the prisoners for whose sake they had been arrested. That such conditions led to closer acquaintance after the war and often kept the men from returning home is so human and understandable that further explanation should be superfluous.

By 7 May, 1947 all remaining former forced labourers, prisoners of war, etc. (in the following called the "actual DPs" or "those remaining in 1945") were "employed in different forms of labour". At the beginning only lumber and land work were open to DPs, but gradually this changed and many DPs followed the country's tendency to industrialism and found employment in various industries.

In the course of the post-war years, a number of DPs left Norway for overseas countries, some also for home.

Norway, however, was far from being the only country with a DP problem. It was in the first place in Germany's West Zone that former prisoners had accumulated. Of the more than 6 million European Jews whose country had been under German occupation and who had themselves been in German concentration camps, only some tens of thousands were left. Very few of them had any wish to return to their "native" country where they had lost everyone and everything. They therefore remained in camps which UNRRA had formed and waited and hoped to go to a country which could offer them an existence worthy of human beings. The same applied to large groups of Polish, Ukrainian and Russian forced labourers.

In 1947, the Norwegian government decided to accept about 500 Jewish refugees from German DP camps as a sort of "compensation" for the 700 Norwegian Jews who had died in German concentration camps. This was the first immigrant transport to Norway. All those who came in it had been in concentration camps.

Many of the above-mentioned 500 Jewish refugees regarded Norway as a transient stage only. "Norway is too near" was the unreflecting comment of the majority, without any of them being able to explain more accurately what they meant by "near" and "far away". In any case, a large number of them left the country again in the course of the first 3–4 years after their arrival.

In 1948 the Communist revolution broke out in Czechoslovakia and consequently a new group of refugees was added to the former ones who still waited in Germany's camps. IRO appealed once more to all the members of the United Nations and asked them to admit groups of refugees. The Norwegian government agreed to accept 150 refugees. The conditions were, however, that work and living accommodation should be obtained before their arrival in Norway. Consequently selection rested only on the working ability and skill of the refugees, and the demand for their particular trades on the labour market of the country.

Later, however, the refugee policy turned in a more charitable direction. Practically all countries were interested only in able-bodied, healthy, preferably skilled immigrants who would not become a burden on public or private social institutions. This resulted in an accumulation of unhappy, doubly miserable and ailing refugees in the German camps. Many were almost desperate after having been screened by one commission after another, only to be rejected and dismissed time after time. Norway then decided to concentrate in the first place on these so-called "hard-core" or "minus" refugees. This applied firstly to blind refugees, and a Norwegian commission selected 40 blind refugees who came to Norway in March, 1950, with their families. In the same year, Norway admitted some doctors and dentists and their families and some few individual immigrants.

The arrangement of a "screening commission" was also kept up later. It proved to be very difficult to place the blind refugees and for many reasons other groups of "minus refugees" were considered, namely tubercular and old people. In 1951, 3 commissions went to Germany and Trieste, and these resulted in the same number of refugee transports to Norway. All these refugees were either tuberculous themselves or relatives of tubercular refugees and therefore cut off from any possibility of emigration to other countries if they did not wish to be separated from their families. The transports included eleven different nationalities and some with no nationality. The placing of these refugees proved very difficult. Numerous organizations helped with this and the State Rehabilitation Institute was also engaged in the task. However the undertaking proved to be almost impossible with Norwegian means only.

In 1952 the Norwegian representative of the Jewish-American Joint Distribution Committee asked the Ministry of Labour for permission to transfer tuberculous Jewish refugees to Norway. The organization was fully aware of the fact that the placing of these refugees in Norwegian employment would be particularly difficult and therefore offered to contribute 1,600 American dollars for every person who could be regarded as a former or present tubercle patient, and 800 dollars for every member of the family who could be considered healthy. AJDC would also pay all expenses connected with the selection of the TB patients and their families, as well as expenses of transport from the respective camps in Germany to Oslo. This appeal which was followed by numerous and lengthy conferences resulted in new screening commissions and transports in 1953 and 1955.

As this brief survey shows, the refugees in Norway are composed of a very special material, made up of different groups very often sharply defined both with regard to war experience, nationality, religion and length of migration. The one thing that is common to them all is that—with few exceptions—they are people with greater difficulties and ailments than an "average refugee population". This does not apply, however, to the former forced labourers who remained in the country.

Without any restriction it may also be said that their experiences during and after the war make it impossible to compare them with "normal" immigrants.

It will be understood from these short sketches that adjustment was not always easy, and that, parallel with the immigration to Norway, there has been a re-emigration to other countries.

This can in no way minify the effort made by Norway for refugee work. The effort is not so remarkable quantitively, but it is concentrated on the worse situated refugees.

2. MIGRATION AND MENTAL DISORDER

In earlier publications Ödegård (1932, 1936), Malzberg (1935, 1935a, 1936) and Malzberg and Lee (1956) especially have studied the incidence of psychoses among migrant populations. Their studies show on the whole that emigration to a certain extent seems to imply a "selection" and usually a negative selection of the population. This leads to the fact, among other things, that the incidence of psychoses is higher among immigrants than among the settled population.

However, it should be stated here that the emigrants studied by Ödegård and Malzberg were for the greater part, *voluntary* emigrants. Even though living-conditions were very bad in those countries from which the "emigrants" came, it cannot be stated without gross exaggeration, that their lives were actually in danger, and emigration was in the last resort a voluntary act, which they could perform or not, as they wished. It is obvious that a "free choice" such as this means many possibilities of motivation. Among these, of course, is the emigrant's personality, reaction pattern, earlier adjustment, ability to cope with existing conditions and so on, all of which are of great, and even perhaps decisive, importance. The situation is totally different for refugees. They have actually lived, more or less, in danger of their lives, their possibilities were very limited, dependent on the willingness or unwillingness of the different countries to receive them. They were grateful to get away anywhere where they did not feel the danger of death so immediately threatening as in their "home country". Under these conditions the theory of emigrant selection is hardly applicable. These considerations refer, in the first place, to political emigrants before the war.

The immigrants who came later came also, *theoretically*, voluntarily, but if we look closer at the conditions under which they lived before emigration to Norway, we see there is little free choice left in the matter. Practically all of them came from the so-called DP camps in Germany, where they either had had to spend many years without really enjoying the freedom they had hoped for at the end of the war, or where they had landed after having fled from their homes, in very real danger of death. Their possibilities of coming to another country were minimal, and the "choice" they had was either to remain in a DP camp in Germany or to come to Norway.

As to the psychiatric literature concerning refugees one refers to *Flight and Resettlement* (1955).

3. THE AUTHOR'S MATERIAL

The present material comprises all persons who came to Norway during or after World War II, until 31 December, 1955, and who have settled here trying to find a new home, either temporary or permanent. Therefore it does not include possible members of diplomatic missions or other persons who could return home if they wished. True some of the patients included in the material went back to their original native country, nevertheless they are included in the material presented, because they either came here, or remained here as *refugees*, they had wished to settle here on an equal footing to the other refugees, and they had not thought of returning to their homelands. Only during their illness did the question of their returning home crop up, either raised by the family at home, or by the treating superintendent, who thought that the patient would be better off in surroundings where he could at least make himself understood. The author saw no reason to exclude these patients from the material. German war criminals are *not* included in the material. They are not refugees and offer totally different problems.

The period covered by this study stretches over 10 years, that is from 1 January, 1946 to 31 December, 1955. (From 8 May, 1945 to 31 December, 1945 no refugees were admitted to Norwegian hospitals, and it is therefore possible to omit this period.) The first task was therefore to clarify the number of refugees in Norway during the observation period. This was no easy undertaking as, in the first place, there is no exact record of the refugees remaining in Norway in 1945–1946. In the second place the Norwegian alien registration office which records all foreigners coming to and leaving Norway, had no special register for refugees. This register was first started in July 1951. Those refugees who arrived or left before this date are included in the large number of tourists, seasonal labourers, artists, etc., who have visited Norway in the course of time, and it has therefore been impossible to trace them.

The author has, therefore, been obliged to apply to the various institutions which have been engaged in work with DPs and later with refugees: The Ministry of Social Affairs, European Aid, The Norwegian Refugee Committee, and the Central Passport Office. The author perused the archives of the governmental institutions which had worked with refugees as well as the files of all the refugee-organizations and the "transport-lists".

By adding up the actual numerical information available about the refugee population of Norway, we get the following results:

TABLE I

Numerical Information on the Refugees in Norway

Date of Count		Total	Source of Information
31.12.1946	abt.	1,000	Ministry of Labour.
31.12.1947	abt.	1,500	Ministry of Labour and transport lists.
1.11.1948	abt.	1,695	Ministry of Social Affairs.
16.3.1949	abt.	1,665	Department for refugees and prison affairs.
10.7.1951		1,773	Central Passport Office.
31.12.1951		2,450	Central Passport Office.
31.12.1952		2,533	Central Passport Office.
31.12.1954		1,903	Central Passport Office.
31.12.1955		1,816	Central Passport Office.

In order to get an "average population" in the 10-year period to be examined here, we have found the arithmetical average total of the information gathered with one total per year.

The totals finally reached by the author are as follows:

TABLE II

Total of Refugees at the End of the Years 1946-1955

31.12.1946	abt. 1,000
1947	abt. 1,500
1948	abt. 1,695
1949	abt. 1,841
1950	abt. 1,978
1951	2,450
1952	2,533
1953	2,074
1954	1,903
1955	1,816
	18,790÷10=1,879

Thus we have reached an "average population" of 1,879 persons. Of these, 60 became psychotic during the observation period, which is 3·19 per cent. of the average population. There are 14 cases of schizophrenias which show an incidence of ·74 per cent., and 42 cases of reactive psychoses with an incidence of 2·29 per cent. These 60 psychotics comprise all post-war refugees who have been treated in Norwegian psychiatric departments or hospitals. The patients were found by tracing the Norwegian Central Register (containing all patients admitted to Norwegian mental hospitals and psychiatric departments) and by inquiring from the superintendents of all those hospitals about any patients who were born outside Norway and who had been admitted to the hospitals. From this—rather comprehensive—material the refugees were traced. The files of the various Norwegian refugee organizations were equally checked. The 51 patients who at the time of the investigation were still in Norway had been personally examined by the author, either in the mental hospitals where they were inmates or in their homes. (Fourteen patients were examined by the author both during their illness and later on in their homes.) Nine patients could not be followed up, 2 were dead and the remainder had left the country. In these 9 cases only the case-histories could be studied.

While the task of obtaining exact totals was very difficult indeed, finding detailed information about the refugee population's composition regarding sex and age was even more so. After the war it was mainly able-bodied men who remained in Norway, and only in later transports, especially in 1947, was a larger number of women included.

By examining the existing information and transport lists the author reached the following results regarding the distribution of sex:

TABLE III

Distribution of Sex in the Refugee Population of Norway

	M.	F.	In All
31.12.1946	abt. 950	abt. 50	abt. 1,000
1947	abt. 1,100	abt. 400	abt. 1,500
1948	abt. 1,275	abt. 420	abt. 1,695
1949	abt. 1,401	abt. 440	abt. 1,841
1950	abt. 1,498	abt. 480	abt. 1,978
1951	1,786	664	2,450
1952	1,818	715	2,533
1953	1,554	520	2,074
1954	1,410	493	1,903
1955	1,362	454	1,816
Total	14,154	4,636	18,790

The 50 psychotic men show an incidence of psychosis of 3·53 per cent., and the 10 psychotic women an average incidence of psychosis of 2·16 per cent.

Information about the distribution according to age has only been available since 1951. It remains on the whole unchanged up to the end of the observation period, and is shown as for 1951 in the following table. As we will see, the distribution according to age is very peculiar, but we draw attention to the fact that it must have been even more peculiar immediately after the war and until the first "hard-core" refugees, among these the "old people transports" were received here in Norway.

TABLE IV

Age Distribution of the Refugees in 1951

Age Group	M.	F.	In All
0– 9	226	219	445
10–19	102	83	185
20–29	150	58	208
30–39	776	135	911
40–49	282	75	357
50–59	153	51	204
60–69	72	30	102
70–	25	13	38
Total	1,786	664	2,450

As it is not possible to reach a totally correct average result regarding the age distribution, we have chosen the 1951 figures as the foundation for further calculations, as this year lies between the extremes 1946 and 1955 both regarding time and, according to the existing information, distribution.

By applying this age distribution to "an average population" that is to say, 1,879 persons (1,415 males and 464 females) we find the following "average age distribution":

TABLE V

Age Group	M.	F.	In All
0– 9	179	153	332
10–19	81	57	138
20–29	119	41	160
30–39	614	94	708
40–49	224	53	277
50–59	122	36	158
60–69	57	21	78
70–	19	9	28
Total	1,415	464	1,879

After these preliminary calculations we have attempted to find the incidence of psychoses classified according to age and the 2 most important diagnostic groups, i.e. schizophrenia and reactive psychosis. In order to ascertain whether the incidence of psychoses among refugee patients was higher than in an average Norwegian matched group, Professor Ö. Ödegård of the Gaustad Mental Hospital was kind enough to calculate the incidence of psychosis that could be expected in an average Norwegian population group of the same size and with the same age and sex distribution as that of the refugee patients.

The calculation is based on the incidence of mental illness in Norway in the period 1946–1950. The results are shown further by the following table:

TABLE VI

Incidence of Psychosis: All Diagnoses

Age Group	No. of Patients	Calculated Incidence	Cases Observed
20–29	119	1·016	13
30–39	614	4·800	16
40–49	224	1·848	17
50–59	122	0·920	2
60–69	57	0·394	2
All males	1,136	8·978	50
20–29	41	0·274	3
30–39	94	0·792	4
40–49	53	0·500	3
50–59	36	0·312	—
60–69	21	0·156	—
All females	245	2·034	10
All refugees	1,381	11·012	60

TABLE VII

Age Group	Number	Incidence of Schizophrenia		Incidence of Reactive Psychoses	
		Calculated	Observed	Calculated	Observed
20–29	119	0·514	5	0·216	8
30–39	614	2·370	6	1·660	9
40–49	224	0·426	3	0·554	12
50–59	122	0·132	—	0·238	2
60–69	57	0·022	—	0·094	1
All males	1,136	3·464	14	2·762	32
20–29	41	0·100	—	0·086	3
30–39	94	0·224	—	0·314	4
40–49	53	0·112	—	0·208	3
50–59	36	0·050	—	0·112	—
60–69	21	0·014	—	0·036	—
All females	245	0·500	—	0·756	10
All refugees	1,381	3·964	14	3·518	42

A comparison between the calculated incidence and the cases observed in the different age groups can hardly be considered of interest with such small totals at our disposal. Tables VI and VII show that the observed incidence for all diagnoses is five times higher than could be expected in a corresponding Norwegian population group. This applies to both males and females. For the schizophrenias alone the difference is somewhat less than five times higher while the reactive psychoses lie far above this number.

With regard to the "actual displaced persons", that is, those who remained behind in 1945, we have attempted to reach an average number by subtracting the arithmetical mean total from the total of DPs in 1946 and 1955, the two extremes on which we have some information.

As on 31 December, 1946 the total was about 1,000 and as on 31 December, 1955 there were 524 actual DPs left (509 males and 15 females). The average is then 764. Of these, 28 became psychotic (27 males and 1 female) which shows a psychosis incidence of 3·61 per cent. With regard to the schizophrenics alone, the incidence is 1·31 per cent and for the 18 reactive psychoses 2·33 per cent.

The author has also attempted to find the age distribution among the actual DPs. Here we have definite information about 524 (509 males and 15 females) only, those who were left as per 31 December, 1955. These can all be reckoned as belonging to a lower age group in 1945, calculated in tens of years.

By applying this age distribution to the DP group in 1945, and besides by calculating the average of the 2 extremes, we come to the age distribution of the "average DP population" of 764 persons.

TABLE VIII

Age Distribution Among the "Actual DP Population"

	1945	1955	Average
–19	77	0	39
20–29	659	40	350
30–39	169	345	257
40–49	71	89	80
50–59	24	37	31
60–69	—	13	7
In all	1,000	524	764

If we compare the incidence of psychosis among the whole refugee population and among the "actual DPs" it proves to be somewhat higher in the latter group. This is due, in the first place, to the remarkable difference with regard to schizophrenia totals, which are ·74 per cent. for all refugees and 1·31 per cent. for the DP group.

The incidence of reactive psychoses is the same in both groups. The relatively high total of schizophrenias among those remaining in the country seems to prove that in this group there have been a number of latent schizophrenias, where the illness may be assumed to have been (co-) decisive for the determination not to return home. The fact that 2 of the remaining 4 schizophrenic patients came into the country as "illegal or accidental" refugees seems to show the same.

While considering these facts, however, one must not forget that a large number of the immigrants who came to Norway after 1945 were also "actual DPs", but remained in Germany instead of Norway. On the whole, the Czechs are the only ones who have fled from their country after the war. The difference in the incidence of schizophrenia can also therefore be explained as being a result of the selection policy which has been upheld by the Norwegian screening commissions. Whilst physical ailments and illnesses were looked upon more or less as a recommendation for immigration to Norway, applicants with mental disorders were usually not considered at all.

Apart from the great "compensation transport" in 1947 (of which so many have re-emigrated), it was first after 1950–51 that the "good-will" (charitably selected) transports came. Before this, 2 "skilled workers" transports came in

1949, that is transports that were not screened but selected according to "trade" qualifications.

The two schizophrenics who were neither "actual DPs" nor illegal immigrants, came with these particular transports, and in both cases they were Czechs who had emigrated a comparatively short time before their arrival in Norway. There were no schizophrenics in any of the transports after 1950. This seems to suggest that the latent schizophrenics who remained in Germany's DP camps in 1945 have gradually become manifest and therefore excluded as possible immigration candidates to Norway. Furthermore, it proved that 10 of the 14 schizophrenics became manifestly psychotic and were hospitalized during the course of the first three years after the war (or, respectively, after their arrival in Norway) and that the 4 who were admitted to hospital after the lapse of these three years showed peculiar traits which may suggest schizophrenic tendencies long before hospitalization proved necessary.

These facts in addition to the course of the illness indicate strongly that the development of the schizophrenic processes seems to be mainly constitutionally conditioned, and that, even in this present small material, schizophrenia, either as a disposition or incipient, seems to be a factor which increases the migration tendency. Ödegård's findings, which, in brief, suggest that there is a connection between the schizoid character (schizophrenia) and emigration, that persons with this special tendency are predisposed to emigration by the way they think, feel and experience social relationships, can also be applied to the present material and may explain some of the schizophrenic psychoses we have found among refugees in Norway. Ödegård supports his theory that the predisposition must be considered of importance for the outbreak of schizophrenic psychoses by stating that the schizophrenia in most cases (almost 80 per cent.) is not manifest in the course of the first 5 years after immigration to U.S.A. He is of the opinion that if the influence of the surroundings is the main reason for the outbreak of the disorder, then this influence would have made itself felt during this period. In our schizophrenic patients we note that almost the opposite is the case, that is, that the schizophrenic disorder breaks out in the majority (11 out of 14) in the course of the first five years. In the author's opinion this is *not* contrary to Ödegård's findings. The assertion regarding the influence of the milieu on the early outbreak of the disease will later be proved in the cases of reactive psychoses. This does not mean that the constitutional, personality factors cannot make their influence felt before the five years have passed. On the contrary, these particular constitutionally conditioned peculiarities will be decisive for the attitude to new conditions met by the emigrant, for the way he attempts to solve his problems, and, also, in most cases, for the unsuccessful result. In our material we also see quite clearly that refugee patients, contrary to Ödegård's, in the initial symptomatology and to a certain extent, can feel the stress of the actual refugee situation. The picture changes very quickly, however. *The symptoms which are brought about by the situation disappear to give way to typical schizophrenic symptoms, totally independent of the refugee existence and its problems:* the schizophrenic process which at first was coloured by the pathoplastic tugging of the actual situation, breaks through, takes the lead and dominates the actual situation both with regard to symptomatology and course.

This observation also explains the contrasts between Ödegård's material and the present one. The external situation, the stress of the milieu, is for our patients so much more frustrating, and encroaches so much deeper into their whole existence and therefore, also has greater influence on the constitutionally conditioned reaction pattern. In all probability it had a greater and more rapid effect on the release of the disorder than the emigrant existence in Minnesota. This explains the early breaking out of the illness in the refugee patients and the symptomatology's pathoplasty, but it says nothing about whether the schizophrenic disorder was *caused* by the external situation. This supposition can be almost totally excluded if we study the case histories and the other considerations described in more detail elsewhere (1958).

The premorbid personality is also of importance in the manifestation of the reactive psychoses, but far from as decisive as in the case of the schizophrenias.

More than 40 per cent. of the reactive states fell ill during the first year, and about 65 per cent. during the first three years after the end of the war (or the refugees' arrival in Norway). Only 8 of 42 had had psychotic reactions before the actual exacerbation. The stresses of the milieu had no doubt a releasing influence in these 8 patients, but considering the background of the personalities mentioned, they can hardly be considered causal. In the other patients, the actual situation, that is the experience of the war and the post-war period, can be traced as causal factors.

In those patients who became ill after a longer period than 3 years, one could however, trace the difficulties of the immigrant situation as decisive for the outbreak of the illness in less than half of the cases. It seems then that the specific difficulties release psychoses mainly in the first 3 years after the refugees have tried to take root. The social and mental hygienic arrangements of the refugees' existence during this period seems therefore to be of the greatest importance. This is described elsewhere in more detail by the author (1958). The late results seem to be mainly the same for refugees as for Norwegian patients suffering from reactive psychoses.

4. Some Remarks on the Aetiology of Mental Disorder among Refugees

The question whether the incidence of mental disease is greater among refugees than among a matched settled average population can be answered in the affirmative, and with a result that leaves no room for doubt. This is in complete agreement with earlier investigations. It is generally assumed that the reasons for this higher incidence is one of the three given below, without a unanimous agreement being reached.

1. A priori, a higher incidence of psychosis in the native land of the refugee.
2. The premorbid personality of the refugees.
3. The mental (and physical) stress during the interval between the uprooting and the outbreak of the disorder.

To answer such a complex problem on the background of an investigation of only 60 psychotic patients would, at first, appear quite hopeless.

The completeness of the material, the personal investigations as well as the author's personal knowledge of the patients' war experiences and original milieu helped to make the task less insoluble.

We may disregard the first of the three above-mentioned possible reasons for the higher incidence. There is no information which can support the assertion that there is a higher incidence of psychosis in the various native countries of the refugees than there is in Norway. Malzberg's investigations, as previously mentioned, show results which refute this assumption.

The author's personal and detailed investigation (1958) has shown that the refugee patients in many ways represent a "minus selection", for example with regard to schooling, socio-economic status, motivation for emigration and so on. This supports the theory of the importance of the premorbid personality. This opinion is strengthened by the fact that the refugees in Norway are of a very specific composition. Besides the forced labourers who remained in Norway in 1945, the refugee population is mainly composed of so-called "minus" refugees, that is, refugees who are disabled or ill, and who were thus "unsuitable" for emigration to other countries. The constitutional foundation for the reaction pattern of these refugees will, because of organic inferiority and the like, be changed and lead to increased vulnerability.

An increased vulnerability such as this will also be brought about by external factors (stress). The present material seems to prove this clearly by the higher incidence of reactive psychoses and by a series of other specific symptoms which we have been able to demonstrate elsewhere (1958).

A careful examination of the case histories and their backgrounds shows clearly that there are mainly two psychodynamic elements which determine the picture of the disease: isolation and feelings of insecurity.

Isolation which occurs when all earlier "group formations" are removed and when the individual is transplanted to a totally strange milieu is here connected with an overwhelming flood of new impressions and stimuli. The lack of ability to digest and absorb the influence of the isolation and overflowing of stimuli results in a total breakdown of the personality. Psychiatrically this phenomenon is manifested by confusional states with disturbances of consciousness.

The feeling of insecurity which is a complex result of both reminiscences of war and post-war experiences, of actual difficulties in the present situation, and of repressed aggression towards every form of "authority", results in doubts in the individual's role-taking and in his relationship to the surroundings. This doubt, together with the projection of personal aggressive feelings, will result in persecutory paranoid delusions. In other cases the lack of security is expressed by jealousy reactions (especially towards partners of another nationality). Also a number of conversion symptoms can be explained as being the result of general insecurity towards the surroundings which can be expected to "accept" somatic ailments, but not mental ones.

All the above-mentioned symptoms and groups of symptoms occur remarkably often in the present material, they stamp, very characteristically, the numerous reactive psychoses, but they are also pathoplastic for the initial stage of endogenous conditioned schizophrenias. We may therefore regard them as dependent on the situation.

The investigation undertaken here does not offer any solution to the problem *either* premorbid personality *or* external stress, but points clearly in the direction of an intense interplay of both factors, of their reciprocal influence and interaction. The result of the present investigation may thus be said to be in agreement with the classical formula in Norwegian psychiatry: Not *either–or* but *both–and.*

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DEATHS ASSOCIATED WITH ELECTROPLEXY

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INTRODUCTION AND PROBLEM

A RECENT court case (16) exposed many of the problems that confront the psychiatrist called upon to perform electroplexy.

In particular it revealed that in 1957, nearly 20 years after E.C.T. had first been used for the treatment of mental disorder, no uniform technique had yet been evolved. The medical witnesses were unable to agree on the use of muscle relaxants or mechanical restraint, but were agreed that there were certain risks attendant upon the use of electroplexy, especially the risk of fractures and the risk to life. It was argued that had the plaintiff received a relaxant drug he might not have sustained bilateral acetabular fractures, but might have suffered an even worse disaster, namely death.

As a result of this controversy, it seemed important to enquire into the death rate associated with electroplexy, and to examine the causes of death, noting especially any possible relationship between death and particular E.C.T. techniques.

(a) *The Death Rate from Electroplexy.* This has been variously estimated by different American authorities, and the results are shown in Table I.

TABLE I

	Number of Deaths	Patient Fatality Rate	Number of Patients Treated	Treatment Fatality Rate
		Per cent.		
Kolb and Vogel (1942) ..	4	0·05	7,207	—
Impastato and Alamansi (1943)	8	0·8	11,000	—
Alexander *et al.* (1956) ..	5	0·1	5,325	1 death in 14,000 treatments
Gaitz *et al.* (1956)	3	0·4	700	1 death in 2,600 treatments

It will be appreciated that no complete data exist regarding the total number of treatments carried out during any particular period of time, so that at the best the above figures relating to patient and treatment mortality rates can only be rough approximations. Oatman (14) stressed that the actual mortality rate may be very different from that suggested by the above figures, because of sampling errors, and because *fatalities* are not always reported. Alexander *et al.* (1) were, however, to some extent aware of this discrepancy, and suggested that the risk of death lies between 1 per 3,000 patients and 1 per 300 patients.

Impastato (8) pointed out that most of the statistics make no allowance for the death rates of patients of different ages. He demonstrated that in 1,000 unselected patients, the most frequent age group receiving E.C.T. was between 30–40, and that the majority of 153 previously reported E.C.T. fatalities had occurred in the age group 50–60. He estimated that the death rate is approximately 0·5 per cent. per patient, in patients over 60, and decreases to 0·033 per cent.–0·025 per cent. in patients between 20 and 30 years of age, but that in "poor risk" patients both these death rates should probably *be doubled.*

(b) *The causes of death* in association with E.C.T. have been tabulated by Impastato (8) based upon a study of 214 previously reported cases together with 40 other cases. He also gives a full bibliography, making further review of the literature unnecessary.

Deaths can be classified into the following four groups:

1. Cardiovascular deaths
2. Cerebral deaths
3. Respiratory deaths
4. Other causes of death including some of the rarities, endocrine disease (Graves' disease, adrenal haemorrhage, etc.), exhaustion, genito-urinary causes (lower nephron nephrosis, uraemia), gastro-intestinal causes (peritonitis, strangulated hernia, rupture of the liver, etc.), leukaemia, death from pre-medication before E.C.T., and cause unknown.

(c) *Possible Relationship of Fatality Rate to E.C.T. Technique.* In Maclay's (13) series, 28 out of 62 E.C.T. deaths had received a relaxant and other premedication, but he stressed that the variety of relaxants and other drugs used made it impossible to ascertain whether any combination was more dangerous than another. Nevertheless he was of the opinion that relaxants might not be an "unmixed blessing" and should be used cautiously in cases with cardiac complications. Kalinowsky and Hoch (11) also believed that the risk was greater with modified than unmodified E.C.T., and it has been stressed that muscle relaxants themselves carry a risk which will be increased when they are followed by E.C.T.

THE QUESTIONNAIRES

In order to ascertain which E.C.T. techniques were in current practice, the first questionnaire was sent to 13 teaching hospitals in London and 42 mental hospitals in June, 1957. The results have been fully reported elsewhere (3) but are briefly summarized below. Questionnaire II was sent in March, 1958, to the same hospitals that were contacted by Questionnaire I. There was nearly a full response to this second questionnaire, which suggests that the subject matter aroused considerable interest.

The following information was requested:

1. A rough estimate of the total number of E.C.T. treatments carried out in the hospital (and associated out-patients' departments) during the years 1956 and 1957. This referred to the number of individual treatments and not courses of E.C.T.

2. The number of deaths that were considered to have been associated with E.C.T. during this period. For the purposes of this investigation only those deaths that had occurred within three days of the last treatment were acceptable, for in cases where this period is longer, it becomes increasingly difficult to incriminate E.C.T. as the direct cause of death.

If there had been any deaths, the hospital was requested to supply full details under the following headings:

Name
Hospital Number
Age
Sex
Psychiatric Diagnosis
Physical Diagnosis
Number of Treatments before Death
Interval between last Treatment and Death
Technique used
Cause of Death
Post-mortem Findings
Any other Remarks

Information about the total number of patients treated was not requested. It was felt that the treatment fatality rate would be a more useful index of the overall position than the fatality rate per patient. It is well known that some patients may die after the first treatment, and that others may have several hundred E.C.T.s before death.

RESULTS

Replies to Questionnaire I showed that no uniform technique of E.C.T. administration was practised by psychiatrists in this country. A survey of the previous literature also revealed no unanimity of opinion.

Briefly, E.C.T. was given modified with thiopentone and Scoline at 60 per cent. of hospitals, and muscle relaxants were used at 85 per cent. Anaesthetists were present for E.C.T. sessions at rather less than 50 per cent. of mental hospitals; at the others treatment was usually given single-handed by the psychiatric registrar, even though thiopentone and muscle relaxants were being used.

Replies to Questionnaire II gave the following results:

1. The number of E.C.T. treatments per annum carried out at a large (2,000 bedded) mental hospital and supporting out-patient clinics ranges from 400 to 8,000.
2. At teaching hospitals in London, the number of E.C.T.s given to in-patients and out-patients ranges from 100 to 2,000 per annum.
3. The total number of treatments carried out during the two-year period at 42 hospitals replying to item I of the circular was approximately 259,000.
4. The number of deaths associated with E.C.T. from all the replies received during this period was 9 (occurring within 3 days).
5. Therefore the incidence of death due to E.C.T. is approximately 1 death in 28,000 treatments, giving a treatment fatality rate of ·0036 per cent.

It is emphasized that the treatment fatality rate may be even lower than this, because some hospitals that failed to provide data concerning the total number of treatments carried out, were able to state that no deaths had occurred, hence the number of deaths is drawn from a slightly larger number of hospitals than those who supplied information about the amount of E.C.T. given.

The fatality rate from E.C.T. taken from over a quarter of a million treatments is very low, far lower than has been recorded by previous authors drawing upon smaller samples.

DISCUSSION ON THE DEATHS REPORTED IN THIS SERIES

Brief descriptions of patients who died following electroplexy are given at the end of this article. Emphasis has been placed upon the circumstances of death together with other relevant findings. The salient points are summarized in Table II.

(a) *Age.* The average age of the series was 67 years. Eight of the patients were aged 60 and over. This indicates that age is an important consideration and must be carefully associated with any figures relating to the E.C.T. fatality rate. This point has been emphasized by Impastato (8) and Eyres (6). Ehrenburg and Gullingsrud (5) reported four deaths in 112 patients aged between 65 and 83 years, though other authorities have reported that E.C.T. can safely be given to elderly patients, and that the incidence of fractures following E.C.T. in feeble senile patients is less than would have occurred during a corresponding period of time from other causes.

(b) *Previous Physical Health.* In only three cases was there no evidence of pre-existing physical diseases. Case I had suffered from a previous coronary thrombosis, and had mild hypertension, dying of left ventricular failure. Case 2 had hypertension, arterio-sclerosis and auricular fibrillation, and gave a history of a recent cerebral thrombosis, yet died of cardiac causes. Case 4 was obese with hypertension and sustained a coronary thrombosis after E.C.T. Case 5 gave a history of cerebral thrombosis 6 months previously and died after a cerebral haemorrhage. Case 6 had suffered for one year with progressive muscular atrophy. Case 7 was obese with hypertension and chronic bronchitis, and died of acute heart failure. These patients all had known physical diseases but had withstood several treatments before death. Muscle relaxants would seem inappropriate in Case 6, who had progressive muscular atrophy. Pre-existing extensive cardiovascular disease could have been clearly diagnosed in many of these cases before they received E.C.T., though only two of the series were considered "poor risk" patients.

(c) *Number of Treatments before Death.* Case 5 died after the first treatment of a second course. Cases 6 and 8 died after "enormous quantities" of E.C.T. had been given. These findings are at variance with those of Maclay (13) who reported that half his series of patients died after the first convulsion.

(d) *The Interval between the last Treatment and Death.* As might have been expected there seemed to be a close association between the mode of death, and the interval that followed the last treatment. Those patients who died of cardiac failure or "shock following E.C.T." either did not recover or died within two hours of the treatment, whereas death from coronary thrombosis or cerebral haemorrhage took place between 30 hours and two days following the treatment.

(e) *Tranquillizers.* Half of the patients in this series were receiving tranquillizing drugs, usually chlorpromazine, at the time of death. It is interesting that all these patients died suddenly of shock or cardiac failure following E.C.T. Weiss (15) reported a patient who suffered a disturbing vasomotor collapse after treatment with E.C.T. while receiving chlorpromazine (450 mg. daily). A marked fall in blood pressure occurred immediately following the treatment, which was not present after subsequent treatments when chlorpromazine was omitted. Weiss postulated that the hypotensive action of chlorpromazine was in this case intensified by E.C.T. Kalinowsky (10) referred to an increase in fatalities among patients undergoing treatment with reserpine and chlorpromazine during E.C.T., and recommended suspending administration of

TABLE II

Case No.	Initials	Age	Sex	Psychiatric Diagnosis	Physical Diagnosis	No. of Treatments Before Death	Interval	Record of Tranquillizers	Technique	Cause of Death
1	E.S.W.	76	F	Senile Psychosis	Hypertension Previous Coronary Thrombosis	4	2 hours	—	Thiopentone and Scoline	Left ventricular failure secondary to hypertension.
2	F.W.	70	M	Agitated Depression	Residual Hemiplegia Arteriosclerosis	3	Minutes	Chlorpromazine 50 mg. t.d.s.	Scoline	Hypertensive cardiac failure accelerated by E.C.T.
3	E.S.	74	M	Depression	—	6	Did not recover	—	Thiopentone Brevidil E.	Myocardial syncope. Coronary artery disease. Emphysema.
4	E.A.L.	73	F	Senile Depression	Obesity Hypertension	3	30 hours	—	Thiopentone and Scoline	Coronary thrombosis.
5	H.A.F.	60	M	Involutional Depression	Previous Cerebral Thrombosis	9 1st of 2nd Course	30 hours	—	Thiopentone and Scoline	Cerebral haemorrhage. Purulent bronchopneumonia.
6	E.C.	62	F	Recurrent Depression	Progressive Muscular Atrophy	Very many	2 days	—	Thiopentone and Scoline	Coronary thrombosis. Progressive muscular atrophy.
7	H.E.B.	60	M	Recurrent Depression	Obesity Hypertension Chronic Bronchitis	5	20 minutes	Chlorpromazine 50 mg. t.d.s.	Thiopentone and Scoline	Acute cardiac failure due to E.C.T. Fatty infiltration of myocardium.
8	M.E.H.	50	F	Catatonic Schizophrenia	—	332	A few minutes	Chlorpromazine 100 mg. t.d.s.	Unmodified	Death due to shock following E.C.T.
9	M.M.	67	F	Schizophrenia	—	5	Did not recover	Chlorpromazine 100 mg. t.d.s.	Unmodified	Cardiac failure. Aortic stenosis.

these drugs for a few days before commencing treatment. Ayd (2) however, took the opposite view, after treating 75 patients without incident suffering from anxiety states with combined chlorpromazine-E.C.T., and regarded this drug as ideal for combination with electroplexy, maintaining that chlorpromazine, acting as a sympathetic depressant, counteracts the excessive sympathetic stimulation that so often accompanies E.C.T. It is possible that the presence of chlorpromazine in the tissues may potentiate the respiratory depression encountered in some patients receiving thiopentone and muscle relaxants, and that unmodified E.C.T. might be a safer procedure in patients receiving this drug.

(f) *E.C.T. Techniques.* It will be observed that only 2 out of the 9 patients who died had received unmodified E.C.T. The majority had received thiopentone and scoline, though Case 2 received scoline alone, and in Case 3, E.C.T. had been modified with thiopentone and suxethonium (Brevidil E). These findings do not in themselves indicate a higher death rate in persons receiving modified E.C.T., but merely reflect the present popularity of modifying techniques. (See results of Questionnaire I.) It is probably a common experience among those who give electroplexy in any quantity to encounter patients who have well tolerated large numbers of unmodified treatments, but whose condition has given rise to alarm following the first E.C.T. modified with a muscle relaxant with or without an anaesthetic. Maclay (13) reported a number of instances of death following the first modified treatment.

Some of these patients may exhibit hypersensitivity to succinylcholine and develop respiratory paralysis, which has been reported on a number of occasions, and recently suxethonium (Brevidil E) has been shown to be not immune from this complication (4). A low serum pseudo-cholinesterase is often found to accompany respiratory difficulties in these patients.

The dangerous properties of muscle relaxants have been stressed by other authors, notably Impastato (8) who reported 5 patients who died following premedication with muscle relaxants *before* they had received E.C.T., and suggested that many of the fatalities attributed to E.C.T. are in fact due to the relaxants.

(g) *Cause of Death and Autopsy Findings.* In the present series there were 2 deaths from coronary thrombosis and one from cerebral haemorrhage which were all subsequently confirmed at autopsy. The remaining cases died fairly suddenly and shortly after treatment, and the cause of death was usually recorded as "acute cardiac failure" or "shock following E.C.T.". In these cases one is at once struck by the meagre autopsy findings, death being presumably due to a severe functional disturbance of delicately balanced physiological mechanisms necessary for life. In some of these patients however there was autopsy evidence of myocardial disease and coronary atheroma which might have contributed to death on some future occasion, and it is in these cases that one may justifiably refer to death as being "accelerated by E.C.T.". The predominant mode of death is cardiovascular—a fact which has been stressed by previous authors.

General Conclusions

1. The risk of death due to E.C.T. is very small and this study shows that it may be even less than previous writers have suggested. It is not, however, an entirely innocuous procedure, to be given without careful consideration of the patient's physical state and psychiatric disorder.

2. The occasional death following E.C.T. necessitates a careful physical examination, with special reference to the cardiovascular system, for all patients, especially those in middle and later life. The day to day physical health of these patients should be carefully checked between treatments.

3. Very great care should be shown to patients in whom technical difficulties have been encountered during previous treatments, and attention directed to changing techniques, generally in the direction of modified to unmodified E.C.T. Additional medication should be reduced to the barest minimum. Electrical modification of the convulsion (i.e. by the Ectonus technique) may be safer than chemical modification in these patients.

4. Nevertheless, there is no evidence from our findings to show that any particular E.C.T. technique carries a lesser risk to life than others.

APPENDIX

Case 1. E.S.W., a female patient aged 76, was admitted on 4 June, 1957 suffering from a senile paranoid psychosis. She had exhibited numerous bizarre unsystematized paranoid delusions for an indefinite period, but her mental condition had deteriorated following treatment for a coronary thrombosis a few weeks previously at another hospital, and she had recently become violent and refused her food. Her physique was described as poor, and she was cyanosed with dorsal kyphoscoliosis and marked emphysema. Her blood pressure was 160/90 with radial arterio-sclerosis. In spite of the physical findings E.C.T. was considered a "justifiable risk".

She received no drugs apart from parentrovite injections after admission. E.C.T., using a current of 30 Joules, modified by thiopentone 0·15 gm. and scoline 35 mg. was given uneventfully at three-day intervals from 26 June. Following the fourth treatment on 5 July, respirations returned, but after 5 minutes they became laboured, and she developed bronchospasm with moist sounds in the chest. She was very restless and did not appear to recover consciousness, showing no response to intravenous injections of aminophylline and nikethamide. She died 2 hours after treatment.

The cause of death confirmed at post-mortem was given as left ventricular failure secondary to hypertension. There was gross pulmonary oedema but this was not regarded as the prime cause of death. There was little atheroma, and the coronary vessels were patent. There were no other lesions of note.

Case 2. F.W., a male patient aged 70, was admitted on 26 January, 1956, suffering from an agitated depression, having made a suicidal attempt by cutting his wrists. He had led an active life as a gardener, but had become depressed following a mild left cerebral thrombosis two months previously, which had reduced his activities. On admission he was emaciated and had a residual hemiparesis. The blood pressure was 160/90 and there was much thickening and tortuosity of the peripheral arteries. The pulse showed an irregular irregularity, but there was no evidence of congestive heart failure. He was seen by a consultant physician and E.C.T. was considered a "justifiable risk". Mentally he was severely depressed and pre-occupied, with ideas of unworthiness, and expressed numerous bizarre hypochondriacal delusions. He was put on chlorpromazine 50 mg. t.d.s. and received two E.C.T.s well modified with 25 mg. scoline on 10 and 14 February, recovery from which was uneventful. Following his third treatment by this technique on 21 February, he became shocked and cyanosed and his pulse was imperceptible at the wrist. Artificial respiration and injections of nikethamide were given without effect, and he died after a few minutes without regaining consciousness. The cause of death ascertained at autopsy was hypertensive cardiac failure accelerated by E.C.T. At the subsequent inquest the verdict was given as "death by misadventure".

Case 3. E.S., a male patient aged 74, was admitted on 8 January, 1957, suffering from a depressive illness with suicidal pre-occupation. A general dealer, he had enjoyed good mental health until some 10 months previously when he developed facial pain for which no cause could be found, and he imagined he had cancer.

On admission he was in poor physical health, but there was no evidence of systemic disease. His blood pressure was 140/80. He received no medication apart from fluids and parenteral vitamins.

On 10 and 11 January he received E.C.T. modified by thiopentone 0·5 gm. and Brevidil E 50 mg. following which it was noted that he took rather a long time to recover. Atropine gr. 1/100 was given ¾ hour before all treatments. On 12 January he was given E.C.T. modified with scoline alone without thiopentone, and recovery was reported as being much quicker. Two further treatments were given on 13 and 15 January but the techniques were not recorded. By 20 January his depression and agitation seemed worse than ever, and he received a further E.C.T. (the 6th) modified with thiopentone 0·5 G and Brevedil E 50 mg. Oxygen was administered both before and after the shock, following upon which he breathed stertorously three times and suddenly expired.

The post-mortem showed myocardial fibrosis, and there was marked narrowing of the descending branch of the left coronary artery due to atheromatous ulceration. The liver was congested but there was no other pathology of note. The cause of death was recorded as cardiac failure due to coronary artery disease and emphysema, and was regarded as accelerated by E.C.T.

Case 4. E.A.L., a female patient aged 73, was admitted on 25 June, 1957 suffering from a senile depressive illness. She was unable to sleep without sedatives, and then woke early and was unable to get off again. She had lost self-confidence and her interest in her friends and her home. She was self-reproachful with suicidal pre-occupation. Her physical health was satisfactory though she was obese and suffered from hypertension, blood pressure 210/110.

She was sedated with sodium amytal gr. 1½ t.d.s. and gr. 3 nocte, but received no other medication. She was given E.C.T., modified with thiopentone and scoline on 1 and 4 July, with no after effects, and some improvement in her mental state was noted afterwards. On 11 July she received a further treatment from which she made a good recovery. Twenty-four hours later she complained of indigestion after supper which was relieved by an antacid mixture. Shortly afterwards she slipped while getting out of her bed, and had to be helped back. She appeared to go off to sleep, but soon afterwards her respirations became stertorous, and she died suddenly at 1.0 a.m. on 13 July. At autopsy the cause of death was shown to be a coronary thrombosis. It was however commented that there did not appear to be any connection between the E.C.T. and the coronary thrombosis.

Case 5. H.A.F., a male patient aged 60, was admitted on 15 January, 1957, suffering from an involutional depression with hypochondriasis. A haulage contractor, he was described as obtuse and mentally retarded, being excessively pre-occupied with his loss of appetite, and imagined that his bowels were "blocked up".

Three years previously he had been treated for a similar depressive illness with 8 E.C.T.s modified with thiopentone and scoline, and had shown a satisfactory response. Six months before admission there was a history of mild confusion and he was thought to have had a cerebral thrombosis at the time.

Physical examination on admission was entirely normal. In spite of the history of a previous cerebral thrombosis, it was decided to give him further E.C.T., particularly as he had shown such a good response to this treatment previously. On 21 January he was given a further treatment modified with thiopentone and scoline, and appeared to recover normally afterwards, and was fully orientated. The following morning he fell out of bed and appeared to have sustained a cerebral haemorrhage from which he died the next day. The interval between E.C.T. and death was 30 hours. The cause of death confirmed at autopsy was cerebral haemorrhage and bronchopneumonia. The pathologists imagined that the latter had been present before the E.C.T. had been given. Extensive atheroma was also present.

Case 6. E.C., a female patient aged 62, was admitted on the sixth occasion in March, 1955 suffering from recurrent melancholia. She complained of insomnia and had much suicidal pre-occupation. She gave a history of having made a number of suicidal attempts previously by various methods. She was first admitted in 1946, and improved temporarily following courses of unmodified electroplexy given without incident. On her last admission her physical condition was satisfactory, though she was mildly hypertensive, blood pressure 150/100.

A year before her death she began to develop weakness and wasting of the lower motor neurone type of her hands, forearms and legs, without sensory changes, which was diagnosed as progressive muscular atrophy. This condition progressed relentlessly, and she was eventually unable to walk, falling several times a day, due to weakness and lack of muscle tone. She became incontinent of urine. She also had bilateral oedema of the ankles which was considered to be due to her neurological condition and not of cardiac origin.

She received no medication during her last admission except sodium amytal given nocte. Since 1956 she had received 23 E.C.T.s modified with thiopentone 0·5 gm. and scoline 50 mg. On her 24th treatment by this technique, she failed to come round and remained cyanosed. A marked tracheal tug was noticed. Following intravenous injections of nikethamide, her condition improved after half an hour, but some 3 hours after E.C.T., she became comatose and her blood pressure fell to 110/60, and she was thought to have had a coronary thrombosis. The following day she appeared better and was fully orientated. Though still slightly cyanosed she was able to sit up in bed and took her food well. The next day, 2 days after treatment, she collapsed and died suddenly. The cause of death confirmed by autopsy was coronary thrombosis. Coronary atheroma and generalized arterio-sclerosis were also noted, together with progressive muscular atrophy.

Case 7. H.E.B., a male patient aged 60, was admitted on 30 November, 1957 suffering from a depressive illness. He had had numerous less severe bouts of depression since 1930, and a year previously had undergone a prostatectomy following which he had never regained his previous health. A clerical worker, he had been depressed with cancerphobia for 5 weeks, and had been unable to sleep properly or concentrate. He repeatedly wished that he was dead, and on the morning of admission made an unprovoked attack on his wife, stating that he wanted to put her out of her misery.

Of pyknic build, he was in fair physical health, apart from a history of chronic bronchitis. His blood pressure was 170/100.

On admission he was put on chlorpromazine 50 mg. t.d.s. which was reduced to 25 mg. t.d.s. on 8 December. He received 5 E.C.T.s all modified with thiopentone 0·25 gm. and scoline 50 mg., atropine gr. 1/100 being given ¾ hour previously. No incidents were reported after the first four treatments, but following the fifth on 12 December, he became cyanosed in the recovery room and was given further oxygen. He died after some 20 minutes had elapsed, and showed no response to oxygen, intravenous and intracardiac nikethamide and artificial respiration.

Autopsy showed moderate hypertensive cardiac hypertrophy, with coronary atheroma and fatty infiltration of the right ventricle. The cause of death was recorded as acute cardiac failure due to electroplexy.

Case 8. M.E.H., a female patient aged 50 at the time of death, was admitted in September, 1937 under certificate suffering from catatonic schizophrenia, having made a suicidal attempt by taking poison and inhaling coal gas. In 1941 her condition deteriorated, and she made frequent unprovoked attacks of violence upon other patients and the nursing staff. She received courses of E.C.T., in 1943, 1946, 1947 and 1948, resulting in temporary behavioural improvement, but unfortunately she tended to relapse, and during these phases became dangerous to persons around her. Maintenance E.C.T. was therefore given up to the date of her death, at monthly, and more recently at fortnightly intervals, when she was also receiving chlorpromazine 100 mg. t.d.s. Between 1943 and her death a total of 332 treatments had been given, and at the time of death she appeared to be in good physical health.

On 14 October, 1957, E.C.T. was given unmodified (as it had been throughout). She came round from the convulsion and was given oxygen. She appeared to be quite comfortable for 2 minutes and was on the point of regaining consciousness when she suddenly collapsed, death occurring in spite of intravenous nikethamide and artificial respiration. The circumstances associated with death were reported to the Coroner who returned the following verdict: "Death due to shock following E.C.T.—accidental death". At autopsy very little abnormal was found. The heart was completely normal and the coronary arteries free from atheroma.

Case 9. M.M., a female patient aged 67, was admitted in December, 1957 suffering from schizophrenia. There was a previous history of episodes of "religious mania", but recently she had been found wandering by neighbours, shouting and referring to herself as "Her Majesty" and had been refusing her food.

On admision her physical health was poor, but there was no evidence of systemic disease. She was very resistant and negativistic. Treatment with chlorpromazine was commenced with a dose of 50 mg. t.d.s. increased to 100 mg. t.d.s. after a few days. She was also given daily injections of parentrovite.

She received four unmodified E.C.T. treatments on alternate days shortly after admission without any untoward effects, though little improvement was noted in her mental state. During the night following one of these treatments she had an attack of what was described as "bronchial asthma" but this subsided after an injection of adrenaline.

On 28 December she received her 5th E.C.T. after which she took only a few breaths. Oxygen, nikethamide, and artificial respiration were given in an attempt to resuscitate her but she died after 10 minutes. All electroplexy had been given unmodified. The cause of death ascertained at post-mortem was cardiac failure due to coronary atheroma and aortic stenosis. The latter had not been detected during life.

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A CONTROLLED COMPARATIVE INVESTIGATION OF THE EFFECTS OF PROMAZINE, CHLORPROMAZINE, AND A PLACEBO IN CHRONIC PSYCHOSIS

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TRANQUILLIZING drugs have become widely accepted as a major form of treatment in psychiatry. However, reports in the literature on these drugs have frequently been conflicting, with a marked tendency for the results of uncontrolled studies to be much more favourable than those obtained in studies where some method of control has been adopted. Sargant (1956) has emphasized many of the social, economic, and moral implications inherent in the extravagant and indiscriminate use of tranquillizers in general and has stressed the need for the proper evaluation of their therapeutic effects. In view of this need, we undertook a controlled comparative study of the effects of two of the phenothiazine compounds, promazine ("Sparine") and chlorpromazine ("Largactil"), in the treatment of chronic psychosis.

METHOD

The principal aims of the investigation were to compare: (*a*) the therapeutic effects of promazine with those of chlorpromazine; (*b*) the effects of these two phenothiazine compounds with those of an inert placebo; (*c*) any toxic effects associated with the use of the different treatments.

For the purpose of the investigation, 63 female patients in a chronic refractory ward of the hospital were selected. These patients were, with a very few exceptions, suffering from some form of chronic schizophrenia characterized by gross disorder of, and disconnection between, thought, affect, and behaviour. Their average age was 57·6 years, and their average duration of illness, 21 years. The usual treatments such as E.C.T., insulin and drugs had failed to effect a cure in these patients who had, in consequence, long since become "institutionalized".

The tablets of the 3 treatments were identical in appearance and were administered on a "double-blind" basis, the hospital dispenser alone knowing to which treatment any particular patient had been assigned at the outset. Group control was used throughout, as opposed to "self" control. The 63 patients were sub-divided, upon a random basis, into 3 equal groups each of which was assigned exclusively to one of the 3 treatments, promazine, placebo, and chlorpromazine. The patients assigned to a particular treatment group received the treatment exclusive to that group throughout the 6 months duration of the trial. The composition of the groups was not made known to the investigators until de-coding at the end of the trial.

For a period of several weeks prior to the investigation each patient, selected for inclusion in the trial, was taken off any treatment, physical or medicinal, which she had been receiving previously. It was intended that promazine, placebo, and chlorpromazine were to be the only treatments to be administered to the patients throughout the trial, which was to extend over a period of 6 months.

Initially it was decided that the patients receiving the phenothiazine drugs should be given a fixed dose of 100 mg. t.i.d. throughout the investigation. The effect of this dosage became apparent on the first day of the trial when an appreciable number of patients suffered from hypotensive attacks and hypersomnia. This complication necessitated the substitution of a phased method of administration of the tablets, so that during the ensuing 2 weeks the dose was increased from 75 mg. daily (25 mg. t.i.d.) of the phenothiazine compounds to a maximum of 300 mg. daily (100 mg. t.i.d.) for the remainder of the trial. The corresponding number of placebo tablets were administered to the control group of patients.

The main criterion of therapeutic response in the patients was to be based upon "improvement scores" derived from the use of the 4 point–52 item Powick Psychiatric Rating Scale. (Full details of the Scale are omitted in the interests of space as it will be the subject of a separate publication. The following summary indicates its scope and structure: Memory—11 items; Perception—4 items; Sleep—3 items; Thought processes—12 items; Affect—7 items; Relationship to Reality—3 items; Behaviour—12 items.) The design of the scale is such that a high gross score may be taken to represent marked abnormality, a low score, indicating relatively slight deviation from the normal. The difference between an initial gross score for a particular patient and a subsequent gross score recorded after a period of treatment may be taken to represent a positive or negative improvement score depending upon the direction of change. Each patient was rated on the scale by a doctor at the commencement of the trial, and this initial gross score remained the standard by which subsequent scores were judged. Each patient was re-rated after 2, 4, and 6 months, that is at 2-monthly intervals throughout the trial. The ratings at the outset, and at the end of each 2-month period, were carried out by 8 different doctors, to each of whom were assigned, upon a random basis, an approximately equal number of patients for rating purposes. Each doctor retained his original set of patients throughout the trial. The mean initial gross scores, determined following decoding at the end of the trial, for the 3 treatment groups, promazine, placebo, and chlorpromazine were 49·9, 49·7, and 50·75 respectively, indicating that the groups were reasonably comparable from the outset.

The nursing staff recorded daily, for each patient, all noisy, aggressive or disturbed incidents which came to their attention during the course of their routine duties. It was hoped that an analysis of these abnormal behaviour incidents, after de-coding at the end of the trial, might be of assistance in differentiating between the therapeutic effects of the 3 treatments in terms of their capacity to modify such behaviour characteristics.

The statistical techniques by which the significance of the results were to be assessed were decided upon in advance and in relation to the numerical data compiled during the investigation. It was intended to subject the rating scores to a series of analyses of variance, and to supplement these by the application of "t" tests as between any two of the three treatments being contrasted. The behaviour incidents recorded throughout the trial were to be subjected to a Chi-square test of significance.

Blood and urine investigations were carried out monthly for each patient, and more frequently in individual cases where this seemed warranted on clinical grounds. A clinical record was kept of any obvious objective symptoms which were manifested by any of the patients during the trial, e.g. pallor, drowsiness, extra-pyramidal symptoms, or other physical abnormalities.

RESULTS

The investigation covered a period of 6 months during which time it was virtually inevitable that some patients would be eliminated for one reason or another. Out of the 63 patients included at the commencement, 51 remained in at the end of the investigation. The reasons for the elimination of the 12 patients who failed to complete the trial were as follows: 6 patients received E.C.T. (administered by the ward doctor who considered this treatment imperative for the patients concerned); 2 patients developed severe extra-pyramidal symptoms (chlorpromazine); 1 patient developed agranulocytosis (chlorpromazine); and 3 patients were withdrawn for miscellaneous physical considerations clearly unrelated to the drug they were receiving (promazine). The loss of patients was equally distributed, as it happened, between the 3 groups, so that the original number of 21 patients in each group was uniformly reduced to 17 by the end of the trial.

Despite the fact that the groups remained numerically comparable, it cannot be denied that some bias was almost certainly effected in the final, as compared with the original, treatment groups, as a result of the loss of these 12 patients. The factor here which, it is considered, exerted the greatest vitiating effect was the elimination of those patients who received E.C.T. These patients constituted, presumably, a particularly disturbed sub-group from the psychiatric point of view, and the withdrawal of 4 of them from the placebo group, as compared with 1 from each of the phenothiazine groups, inevitably reduced to some extent the "random" quality or comparability of the original 3 groups. The distribution of E.C.T. between the 3 groups might further suggest that the phenothiazine compounds were exerting a greater damping down or "tranquillizing" effect than the placebo, but the numbers concerned are, however, too small for any reliable deductions to be based upon them.

The therapeutic results have been presented, summarily, in the series of Statistical Tables, I–XII. Certain discrepancies may be apparent in these Tables with regard to the anticipated as compared with the actual number of observations recorded, and which are reflected in the "degrees of freedom" used. These discrepancies were a function of 2 factors: (*a*) partial and periodic failure on the part of individual doctors, for various reasons, to carry out ratings on some of their allotted patients at the specified times; (*b*) elimination, as the trial progressed, of 12 patients for reasons already given.

TABLE I

Analysis of Variance
(Rating Scale Improvement Scores Recorded after 2 Months)

Source of Variation	D of F	S of S	Variance	F	P	
Between doctors ..	5	722·4	144·5	1·5	> ·05	NS
Residual	30	2949·9	98·3			
Totals	35	3672·3				

Variance for "Between Treatments" and Variance for "Treatment × Doctor interaction" were each found to be less than the "Residual" into which, consequently, their degrees of freedom and sums of squares have been pooled in the above Table.

TABLE II

t-tests Between Mean Improvement Scores Recorded after 2 Months

Treatments	M_1 – M_2	$S.E._D$	*t*	D of F	P	
Promazine-Placebo ..	(1·0)–(−2·9)	3·5	1·1	22	> ·05	NS
Promazine-Chlorpromazine	(1·0)– (2·0)	4·4	0·2	25	> ·05	NS
Placebo-Chlorpromazine ..	(−2·9)– (2·0)	4·8	1·0	19	> ·05	NS

TABLE III

Analysis of Variance
(Rating Scale Improvement Scores Recorded after 4 Months)

Source of Variation	D of F	S of S	Variance	F	P	
Between treatments ..	2	549	274·5	1·5	> ·05	NS
Between doctors ..	7	2162·1	308·9	1·7	> ·05	NS
Residual	34	6042·1	177·7			
Totals	43	8753·2				

Variance for Treatment × Doctor interaction less than Residual (cf. footnote, Table I).

TABLE IV

t-tests Between Mean Improvement Scores Recorded after 4 Months

Treatments	M_1 – M_2	$S.E._D$	*t*	D of F	P	
Promazine-Placebo ..	(−0·7)–(−6·5)	5·4	1·1	28	> ·05	NS
Promazine-Chlorpromazine	(−0·7)– (2·6)	5·4	0·6	30	> ·05	NS
Placebo-Chlorpromazine ..	(−6·5)– (2·6)	4·8	1·9	24	> ·05	*

* Difference between Placebo and Chlorpromazine may be said to approach significance at 5 per cent. level of confidence (required t-value = 2·06), with ·10 > P > ·05 at least suggesting possible superiority of Chlorpromazine.

Reference to Tables I and II will reveal that statistically speaking, there was no significant difference between the therapeutic effects of the 3 treatments in terms of the rating scale improvement scores at the end of the first 2 months of the trial. None of the sources of variation considered in Table III, and relating to the improvement scores after 4 months, provide any significant differences. On the other hand, it may be seen from Table IV that there is a difference between the mean improvement scores for the placebo and chlorpromazine, at the end of 4 months, which may be said to approach statistical significance, and at least suggesting the possible superiority of chlorpromazine.

TABLE V

Analysis of Variance
(Pooled Improvement Scores Recorded after 2 and 4 Months)

Source of Variation	D of F	S of S	Variance	F	P	
Between treatments ..	2	638·4	319·2	2·3	$> \cdot 05$	NS
Between doctors ..	7	2206·8	315·3	2·3	$< \cdot 05$	S
Residual	70	9628·8	137·5			
Totals	79	12474				

Variance for Treatment × Doctor interaction less than Residual (cf. footnote, Table I). Differences between Doctors are significant at 5 per cent. level of confidence.

TABLE VI

t-tests Between Means Derived from Pooled Improvement Scores Recorded after 2 and 4 Months

Treatments	$M_1 - M_2$	$S.E._D$	t	D of F	P	
Promazine-Placebo ..	(0·1)–(−5·0)	3·4	1·5	52	$> \cdot 05$	NS
Promazine-Chlorpromazine	(0·1)– (2·3)	3·5	0·6	57	$> \cdot 05$	NS
Placebo-Chlorpromazine ..	(−5·0)– (2·3)	3·5	2·1	45	$< \cdot 05$	S

Difference between Placebo and Chlorpromazine is significant at 5 per cent. level of confidence, reflecting the superiority of Chlorpromazine.

TABLE VII

Analysis of Variance
(Rating Scale Improvement Scores Recorded after 6 Months)

Source of Variation	D of F	S of S	Variance	F	P	
Between doctors ..	7	3731·1	533·0	4·7	$< \cdot 01$	S
Residual	34	3823·9	112·5			
Totals	41	7555				

Variance for Between Treatments and Variance for Treatment × Doctor interaction each less than Residual (cf. footnote, Table I).

Differences between Doctors are significant at 1 per cent. level of confidence.

TABLE VIII

t-tests Between Mean Improvement Scores Recorded after 6 Months

Treatments	$M_1 - M_2$	$S.E._D$	t	D of F	P	
Promazine-Placebo ..	(2·0)–(−1·6)	5·8	0·6	27	$> \cdot 05$	NS
Promazine-Chlorpromazine	(2·0)– (2·0)	5·0	0·0	28	$> \cdot 05$	NS
Placebo-Chlorpromazine ..	(−1·6)– (2·0)	4·7	0·8	23	$> \cdot 05$	NS

When the improvement scores at the end of 2 months are pooled with those at the end of 4 months, as in the Analysis of Variance shown in Table V, the differences between doctors are found to be significant at the 5 per cent. level of confidence; and the "t" value for the difference in means between these pooled improvement scores for the placebo and chlorpromazine is significant at the 5 per cent. level, reflecting the superiority of chlorpromazine (cf. Table VI). From Tables VII and VIII it may be seen that there were no statistically significant differences between the 3 treatments in terms of the improvement scores recorded at the end of 6 months; but the differences between doctors are significant at the 1 per cent. level of confidence. When the

TABLE IX

Analysis of Variance
(Pooled Improvement Scores Recorded after 2, 4 and 6 Months)

Source of Variation	D of F	S of S	Variance	F	P	
Between treatments ..	2	679·9	339·95	2·6	$>{\cdot}05$	NS
Between doctors ..	7	4839·5	691·4	5·3	$<{\cdot}01$	S
Residual	112	14569·6	130·1			
Totals	121	20089				

Variance for Treatment × Doctor interaction less than Residual (cf. footnote, Table I). Differences between Doctors significant at 1 per cent. level of confidence.

TABLE X

t-tests Between Means Derived from Pooled Improvement Scores Recorded after 2, 4 and 6 Months

Treatments	$M_1 - M_2$	$S.E._D$	t	D of F	P	
Promazine-Placebo ..	(0·7)–(−3·7)	3·2	1·4	81	$>{\cdot}05$	NS
Promazine-Chlorpromazine	(0·7)– (2·2)	2·9	0·5	87	$>{\cdot}05$	NS
Placebo-Chlorpromazine ..	(−3·7)– (2·2)	2·7	2·2	70	$<{\cdot}05$	S

Difference between Placebo and Chlorpromazine is significant at 5 per cent. level of confidence, reflecting the superiority of Chlorpromazine.

TABLE XI

Incidents of Disturbed Behaviour, Recorded Throughout Total Trial Period of 6 Months

Treatment	Patients Disturbed 0–5 Times	Patients Disturbed 6–10 Times	Patients Disturbed 11–15 Times	Patients Disturbed 16–20 Times	Patients Disturbed 20+ Times	Total
Promazine ..	13	0	1	0	3	17
Placebo ..	8	3	1	2	3	17
Chlorpromazine	13	2	1	0	1	17
Total ..	34	5	3	2	7	51

For purposes of Chi square test the numbers of patients appearing in Columns 2–5 are too small for reliability; the Table has therefore been compressed as below.

TABLE XII

Incidents of Disturbed Behaviour for Whole Trial

Treatment	Patients Disturbed Less than 6 Times		Patients Disturbed More than 6 Times		Total
	Observed	(Expected)	Observed	(Expected)	
Promazine ..	13	(11·3)	4	(5·6)	17
Placebo	8	(11·3)	9	(5·6)	17
Chlorpromazine ..	13	(11·3)	4	(5·6)	17
Total ..	34	(34)	17	(17)	51

$\chi^2=2{\cdot}83$, df 2, $P>{\cdot}05$, NS.
NS=Not statistically significant. S=Statistically significant.

improvement scores at the end of each 2-month period of the trial, i.e. after 2, 4, and 6 months, are pooled together, the Analysis of Variance (cf. Table IX) indicates differences between the doctors which are significant at the 1 per cent. level of confidence; whilst the "t" value (cf. Table X) for the difference between the placebo and chlorpromazine, in terms of these combined scores, is significant at the 5 per cent. level of confidence, reflecting the superiority of chlorpromazine. Tables XI and XII present an analysis of the frequency of disturbed behaviour incidents recorded daily throughout the trial; however, the differences between the treatments, in terms of those incidents, are not statistically significant.

No serious side-effects were associated with the use of promazine or the placebo, but chlorpromazine caused agranulocytosis in 1 patient, and severe extra-pyramidal symptoms in 2 others. The results of the repeated laboratory investigations were essentially negative for the 3 preparations, with the obvious exception of the single case of agranulocytosis.

Discussion

It has already been observed that there were no statistically significant differences between the improvement scores for the 3 treatments, as analysed together or in pairs, at the end of the first 2 months of the trial. This is perhaps rather surprising in view of the fact that Elkes and Elkes (1954), and more recently Crawford Little (1958), reported that the effects of chlorpromazine became apparent within a period of weeks rather than months. (It is necessary to state here, however, that Crawford Little did not obtain statistically significant differences between the effects of chlorpromazine and a placebo in his investigation.) Some possible explanation of the negative findings in this trial after 8 weeks might be: (*a*) that the specific therapeutic effects of chlorpromazine and promazine, if any, were too slight to be revealed by the type of scale used, and/or that the numbers of patients in each group were too small for any differences to reach a statistical level of significance; (*b*) that the maximum effects of the phenothiazine treatments having been exerted in, say, the first 2–4 weeks, had already begun to wane after 8 weeks, or that, in contradistinction to the findings of other workers, 8 weeks is not sufficient time to allow for the maximum effects to manifest themselves; (*c*) that if, during the first 8 weeks, several sets of ratings had been carried out, then the pooled scores for the successive ratings might, cumulatively, have provided significant results; (*d*) that the effects of the 2 phenothiazine treatments were so comparable in terms of improvement scores that in the analysis of variance they mutually diluted their respective differences from the placebo, resulting in a deceptively negative result; the negative t-tests between the separate pairs of treatments, however, would seem to rule this out as a plausible explanation. It may be interesting to speculate about these points but further research is necessary to elucidate them. All that can be stated with any degree of confidence is that the improvement scores at the end of 2 months provided insufficient statistical grounds for rejecting the null hypothesis, namely, that there were no real differences in effects between the 3 treatments.

The differences between treatments at the end of 4 months were again found to be not significant in the relevant analysis of variance. However, the t-statistic for the difference between chlorpromazine and the placebo approaches significance, with the P value nearer to the 5 per cent. than the 10 per cent. level of confidence. This may be taken at least to suggest a possible superiority of chlorpromazine over the placebo (chlorpromazine mean improvement score, 2·6; placebo mean improvement score −6·5). It is necessary to mention here that there may be doubts, entertained by some statisticians, as to the validity of carrying out t-tests, as between pairs of treatments, when the differences between all treatments used are not found to be significant in the analysis of variance.

Speculation has already been entertained, in relation to the negative findings for the first 2 months of the trial as to the possible cumulative effects of pooling sets of improvement scores recorded on successive occasions. When this procedure is adopted for the scores obtained at 2 months and again at 4 months, significant differences are found to obtain, at the 5 per cent. level of confidence, between the doctors themselves and between chlorpromazine and the placebo. The differences between doctors may be reliably deduced from the analysis of variance, and are interpreted as a salutary reminder of the relative and personal nature of observations in general, however "objective" they may superficially appear to be. The superiority of chlorpromazine over the placebo, suggested already by the analysis of the scores at the end of 4 months, would appear to be confirmed by the t-statistic obtained from the pooled scores concerned.

The set of scores recorded at the end of 6 months reveal, in the analysis of variance, that the differences between doctors are significant at the 1 per cent. level of confidence. It might not be unreasonable to interpret this as an indication that the trial results are now telling us, however non-specifically, as much about the doctors as about the treatments they are attempting to evaluate. The former superiority of chlorpromazine over the placebo is now no longer in evidence, and this would seem to be due to 2 factors: (*a*) the high negative mean improvement score of $-6 \cdot 5$ recorded for the placebo group after 4 months has been reduced to $-1 \cdot 6$; (*b*) the corresponding positive mean improvement score for chlorpromazine after 4 months has been reduced from 2·6 to 2·0. A tentative interpretation here might be that most of the placebo patients progressively relapsed somewhat from their status at the outset during the ensuing months, reaching a nadir of deterioration after some 4 months, following which there occurred some degree of restoration to their former state.

The procedure of pooling and analysing together the 3 sets of improvement scores, obtained at 2, 4, and 6 months respectively, furnishes little additional information. The differences between doctors, for what may be described as the whole trial, are found to be significant at the 1 per cent. level of confidence in the analysis of variance; and the superiority of chlorpromazine over the placebo is demonstrated at the 5 per cent. level by the t-statistic.

It may be said then, that the trial failed to differentiate between the treatment effects, in terms of the rating scale, of promazine and the placebo; whereas it did differentiate between chlorpromazine and the placebo, and favoured the former rather than the latter. If the results, and their interpretations, be accepted as evidence of the superiority of chlorpromazine over the placebo, the question then arises as to *how much* superior. This is a question to which no unequivocal answer can be given. In general, nevertheless, it may be said that the effects of chlorpromazine were in no way dramatic or curative. The largest mean improvement score recorded for the chlorpromazine group was 2·6 (rating after 4 months), and this represents a most meagre therapeutic gain in a group with an initial gross score of 50·75. On the other hand the largest difference in mean improvement scores, between the chlorpromazine and placebo groups was 9·1 (after 4 months), which may reflect to some extent, more fairly the therapeutic performance and superiority of chlorpromazine.

One possible and extenuating explanation of the therapeutically ineffectual performance of promazine in this trial might be that it is less potent, dose for dose, than chlorpromazine, and, in addition, that 300 mg. daily is inadequate to obtain its possible optimal effects in chronic psychotics. Barsa and Kline (1957), reporting upon a comparative study designed to evaluate the effect of promazine as compared with chlorpromazine in chronically disturbed schizophrenic female patients, concluded that "promazine appears to have a chlorpromazine-type of clinical effect. However, in 9 of the 21 patients, it required double the amount of promazine to equal the clinical effect of chlorpromazine." Some of their patients received a dosage of promazine as high as 300 mg. q.i.d.

It may be thought surprising that the analysis of the incidents of disturbed behaviour recorded throughout the trial, did not reveal any superiority of either of the phenothiazine compounds over the placebo. Perhaps the comparatively small number of patients in each group, and the withdrawal of 4 patients who required E.C.T. from the placebo group, and compared with only 1 from each of the other groups, were operative factors here. However, Crawford Little (1958), and Mitchell (1956) obtained similarly negative results for chlorpromazine as a behaviour-modifying drug, and Sibilio *et al.* (1957) reported negative results with promazine, so that one might be justified in entertaining a certain degree of scepticism concerning the claims so often made for these compounds as "tranquillizers".

It is necessary to emphasize in relation to the rating scale as a measurement of therapeutic response that some items are mutually exclusive, e.g. those for over- and under-activity, so that an improvement in one direction may be cancelled out by a deterioration in the opposing direction, with little or no change in the relevant gross score for the patient concerned. Furthermore, specific gains effected in relation to a small number of items may be so greatly diluted by the large number of remaining and unaffected items that their presence and significance may not become apparent in the relevant statistical analyses. An item analysis suggests itself as a possible means of dealing with these difficulties and a method by which specific, as distinct from general, effects of the different treatments might be identified. Further research into this aspect of the investigation may be undertaken at a later date.

The case of agranulocytosis attributable to chlorpromazine is a further reminder of a very real and serious danger attendant upon the use of this drug. It must be borne in mind however, that the use of promazine has also been associated with the same grave condition, and one fatal outcome has been reported by Woodward and Solomon (1956), and another by Earle (1957).

SUMMARY

A comparative study of the effects of promazine, chlorpromazine, and a placebo in chronic psychosis is reported upon.

Sixty-three chronic psychotic in-patients were sub-divided, on a random basis, into 3 equal groups each of which was assigned exclusively to one of the 3 treatments, promazine, chlorpromazine, and placebo, for a period of 6 months.

The treatments were administered orally, and upon a "double-blind" basis. The dosage of the phenothiazine drugs was increased to a maximum of 300 mg. daily within the first 2 weeks of the trial, and was sustained at this level thereafter.

The therapeutic response of the patients was based, in the main, upon improvement scores derived from a comprehensive 4 point–52 item psychiatric rating scale. Eight doctors carried out the ratings on the trial patients at the outset, and subsequently at 2, 4 and 6 months respectively.

Statistical analysis of the results provided some evidence, at the 5 per cent. level of confidence, that chlorpromazine was superior in its therapeutic effects to the placebo. Though manifest, the superior effects of chlorpromazine, when interpreted in terms of the rating scale as a whole, were deemed to be meagre in the extreme. No significant differences were found to obtain between promazine and the placebo. A significant difference, at the 1 per cent. level of confidence, was discerned between the ratings of the different doctors participating in the investigation, and a comment was made as to the possible implications of this phenomenon.

Incidents of disturbed behaviour, relating to the trial patients, were recorded by the nurses throughout the trial, but these did not help to differentiate between the therapeutic effects of the 3 treatments.

One case of agranulocytosis was associated with the administration of chlorpromazine, recovery occurring upon discontinuance of the drug and the application of prompt remedial measures.

It is concluded that whatever may be the merits of promazine and chlorpromazine in other psychiatric or medical fields, these phenothiazine compounds are not very effective in the treatment of chronic psychosis. Chlorpromazine would appear to be more potent than promazine and, slight though its effects may be, it must be remembered that any drug which may facilitate the management or rehabilitation of chronic psychotics should not be lightly discarded pending the discovery of more effective methods of treating these unfortunate members of our society.

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A CHILDREN'S APPERCEPTION TEST—AN INVESTIGATION OF ITS VALIDITY AS A TEST OF NEUROTICISM

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INTRODUCTION

Statement of the Problem

AN examination of recent catalogues of tests published by the Australian Council for Educational Research, the Psychological Corporation of America and the National Foundation for Educational Research, has shown that few well-standardized and validated tests of personality are available for use with children.

In the adult field there has been more activity, and many researches have investigated the value of ratings, questionnaires and inventories, objective behaviour tests, constitutional and physiological measures and projective techniques for the assessment of personality. Eysenck (1953) has suggested that much of this work is wasted because it was not designed to test any particular hypothesis. He suggests that an initial requirement is the construction of a mathematical model of personality organization. Measurement in the field of personality, he insists, is impossible until the dimensions along which such measurement can take place are known.

The present experiment is an attempt to relate the results of large-scale factorial studies of personality in the adult field, to the problem of personality measurement in children. It also attempts to construct an objective test for use with children, measuring a major dimension of personality (neuroticism), isolated as a result of these factorial studies.

This approach was suggested because factorial studies of adult personality appeared to have been conducted with much more scientific vigour than many of the studies with children and also appeared to suggest more definite hypotheses. Secondly, these studies having suggested a number of dimensions of personality, it seemed profitable to construct a test based on adult criteria of one of these (neuroticism) and to determine the discriminatory power of the test by comparing its results with external criteria of maladjustment in children. If significant differences were found it might suggest greater confidence in making further predictions in the field of personality measurement in children, based on work conducted in the adult field. This approach was encouraged in that several studies with children had produced results compatible with the results from factorial studies with adults (Webb, 1915; Maller, 1934; Cracknell, 1939; Brogden, 1940).

In the introduction to "Dimensions of Personality" Eysenck (1947) stated that the aim behind his work was, ". . . to discover the main dimensions along which . . . measurement of the emotions can take place, and to provide experimental evidence of the feasibility of such measurement". He planned and carried out a large-scale factorial study on the intercorrelations of 39 trait ratings, whose presence or absence in 700 male neurotic soldiers was assessed by psychiatrists. These items covered the social history of the patient, his personality, and his symptoms. The full table of 741 intercorrelations is contained in Eysenck (1944).

Two main factors were discovered—a general factor of "neuroticism" and a bipolar factor of introversion–extraversion. The first major factor of neuroticism was characterized by: "Badly organized personality, dependent, abnormal before illness, boarded out, narrow interests, little energy, abnormality in parents, schizoid, dyspepsia, poor muscular tone, unsatisfactory home, no group membership, and cyclothymic". These items he considered to delineate, "a general lack of personality integration, lack of adaptability, and lack of general drive which might justifiably be called 'neuroticism'."

There is collateral evidence in the literature to suggest that this factor of neuroticism applies also to children. Webb (1915), using Spearman's factorial methods on correlations between a variety of ratings, carried out a study on 200 students and 120 children. Traits such as strength of will, excellence of character, perseverance in the face of obstacles and trustworthiness all tended to go together. These qualities seem to characterize the opposite pole of the "neuroticism" factor. He called this factor "W" (Will). Brogden (1940) confirmed the existence of the "W" factor by a factorial-analysis of the intercorrelations between a series of experimental tests. His results showed that this factor was associated with "resistance to suggestion". Cracknell (1939) tested 103 10-year-old school children and similarly showed significant correlations between positive personality traits and resistance to suggestion. It will be remembered that, "Suggestibility was found to be strongly correlated with neuroticism, both when comparing neurotics with normals, and when comparing the more seriously ill with the less seriously ill it was found that the degree of neuroticism was reflected in the suggestibility scores (Eysenck, 1947)". Maller (1934) also isolated a factor "C", or readiness to forgo an immediate gain for the sake of a remote but more important gain. He analysed the intercorrelations of tests of honesty, co-operation, inhibition, and persistence applied to some 700 pupils. Both Webb's "W" and Maller's "C" factors appear to correspond closely to Eysenck's first general factor of neuroticism and to suggest that measurement of this factor in children would appear to be more rational and systematic than the construction of tests, unrelated to any sound theoretical framework of personality organization.

As suggested earlier, however, there is no test known to the author which attempts a valid and quantitative measurement of neuroticism in children (neuroticism defined operationally as a result of experiment) and which is related to a theoretical framework of personality organization, developed as a result of lengthy programme research.

Suggestion for Solution of Problem

There are two techniques which are used almost exclusively in the practice of personality testing. These are the projective and questionnaire methods.

Although Bell (1948) has made several major criticisms of projective techniques, several studies have shown the diagnostic value of projective techniques, particularly for example the careful experimental work of Cox (1951) and Sen (1950). Cox gave the Rorschach test to two groups of normal and neurotic children equated for age and intelligence. Twenty-six Rorschach scoring categories were intercorrelated for the whole group of 120 children. Five factors were extracted. The second major factor was interpreted as one of neuroticism. Its highest loading was in the normal-neurotic dichotomy (.73). This finding was similar to that reported by Sen (1950). He used 100 Indian students, 60 men and 40 women. Three factors were extracted, one of which was neuroticism. This factor correlated .685 with ratings for neurotic tendencies.

Validation studies of projective techniques appear to have demonstrated their value in the assessment of adjustment, or of clear-cut diagnostic categories. Studies which have tried to demonstrate the value of these techniques in the assessment of personality-traits have been less successful. A review of the available evidence on this point is unfortunately not possible in the present instance. Possible reasons for doubting the value of the projective approach are nevertheless the lack of a theoretical framework for the tests, the fact that they do not rest on clear-cut assumptions and have not given rise to any satisfactory theory of personality.

Some advance has also been made in the dimensional analysis of adult personality and in its measurement, particularly by questionnaire methods.

Excellent summaries of the value and validity of questionnaires have been given by Ellis (1946) and Ellis and Conrad (1948). Although personality inventories and questionnaires have several advantages such as ease of administration and scoring and a standardized form, there are several serious disadvantages. Faking and the self-halo effect are important problems. In addition, questions are often artificial, suggestive and dependent on cultural factors.

Both Vernon (1938) and Eysenck (1953) have suggested, that whilst it was probably justifiable to conclude that questionnaires measured psychologically significant variables, at the same time the testees *must be adequately motivated to give candid responses.* Ellis (1953) concluded that questionnaires were easily faked and did not usually give significant group discriminations when used with vocational, academic, socioeconomic, disabled or ill groups.

It would appear distinctly relevant to inquire into the results achieved by questionnaires administered and validated *under suitable conditions.* Typical of questionnaires used in this connection is the Maudsley Medical Questionnaire. It consists of questions dealing largely with physical complaints, often made by neurotic subjects. The reason for constructing the questionnaire in this form lay in the fact that people are more forthcoming when questioned about bodily health. The test was administered to 1,000 normal and 1,000 neurotic members of H.M. Forces. It tended to show high correlations with psychiatric opinion. In one study a correlation of +.70 was obtained, although the psychiatrist was unaware of the questionnaire results (Eysenck, 1947). The test was also shown to discriminate between the more seriously ill and the less seriously ill neurotics.

Equally impressive differences between neurotics and normals have been achieved by Crown (1948) using his Word Connection List. This test, which is an "alternative choice" version of the word association test, provides two responses which are offered to each stimulus word. The score is the number of neurotic responses underlined. The importance of this test, like the Maudsley Medical Questionnaire, lies in its ability to demonstrate significant differences in test responses between neurotics and normals and not to put the patient on his guard.

Apart from the Maudsley Medical Questionnaire there are three other measures in current use with adults which have been reported to be good measures of neuroticism, though each is composed of items referring directly to the mental health of the patient. The first is the Taylor Scale of Manifest Anxiety (Taylor, 1951, 1953, 1956), a questionnaire especially constructed from items of the Minnesota Multiphasic Personality Inventory. This scale and its intercorrelations have been reviewed by Franks (1956). It correlates highly with the Maudsley Medical Questionnaire and the "Psychasthenia" scale of the Minnesota. Franks obtained a correlation of +.92 between the Taylor Scale and the Maudsley Medical Questionnaire. A further measure of neuroticism is the Maudsley Personality Inventory (Eysenck, 1956).

Several suggestions have emerged with regard to the development of an objective measure of neuroticism for use with children.

1. Some ten years of programme research at the Institute of Psychiatry has resulted in the isolation of two main factors or dimensions of personality. One of these has been identified as "neuroticism". This dimension has been defined operationally. Several experiments have been quoted which suggest that this same dimension applies also to children.

2. Projective techniques appear to be of value in discriminating between different diagnostic groups. There are nevertheless many difficulties attached to projective testing. One of the main advantages of the method is that it puts people less on their guard. Questionnaires largely lack this quality. Questionnaires were nevertheless seen to discriminate reliably between normal and neurotic groups, providing the questions did not refer too obviously to the mental health of the subject.

3. When given under suitable conditions questionnaires appear to produce discriminating scores. As it is often difficult to approximate these "suitable conditions" and to obviate the frequent tendency of people to fake their answers, the suggested approach might be to combine the projective and questionnaire methods of assessment. The scoring would then be in accordance with neuroticism–questionnaire items and so also be related to a theoretical background of personality organization. Scoring would also be objectified. Defensiveness would presumably be less. Selection of different items of "significance" in the stories, according to the theoretical bias of the examiner, would not be applicable.

The present experiment was designed to check the suggestion that a combination of the projective and questionnaire approaches might be of value in developing a valid test of neuroticism for use with children.

Design of Experiment

Material

A new series of projective plates relating to an English culture pattern was constructed. Eleven situations were arbitrarily chosen to represent a cross-section of situations which might provide responses which could be measured in terms of the normal-neurotic dimension.

The original plan was to use both boys and girls in the study. This was later abandoned to make the study a more comprehensive one for boys only and to discover whether further work should be done in validating the test on other groups of boys and girls (an equivalent set of plates had already been constructed for girls).

Seven of the eleven plates contained no reference to girls (1M, 2M, 3M, 4M, 5M, 6M, 7M), whilst the remaining four plates showed both boys and girls (8MF, 9MF, 10MF, 11MF).

Each of the eleven situations was first described by the writer and a qualified artist asked to reproduce these situations in black and white ink drawings. A new method of scoring projective material was then devised based on four questionnaires of adult neuroticism (Maudsley Medical Questionnaire, Maudsley Personality Inventory, Taylor Test of Manifest Anxiety and the Psychasthenia scale (M.M.P.I.). There was some overlap in the questions from these scales. In order to avoid unnecessary duplication, a new scale was constructed to contain as far as possible relatively "pure" items. Twenty-two variables were isolated and included in the final scoring form. Definitions of these twenty-two scoring items are contained in "Design of Experiment—Scoring".

Population

Two groups of boys were used. The first group consisted of 328 "normal" boys between the ages of eleven and fourteen who were attending Grammar and Secondary Modern Schools in St. Helens and Manchester. The second group was composed of 47 maladjusted boys who were full-time resident students at the Chaigeley School for maladjusted children (age range 11-16).

The "normal" sample was selected on the basis of the distribution of Secondary Modern to Grammar School boys in St. Helens during 1957. The figures applied to eleven and twelve year olds. There were 1,753, of which 15·5 per cent. entered Grammar School and 6·8 per cent. the Secondary Technical School. The remainder attended Secondary Modern Schools. There were thus 22·3 per cent. Grammar and Technical and 77·7 per cent. Secondary Modern students.

An attempt was made to construct the present sample with regard to these percentage distributions.

In the present sample of 328 boys, 80 were Grammar School students whilst the remaining 248 were attending Secondary Modern Schools. Approximately 24 per cent. were thus attending Grammar Schools and 76 per cent. Secondary Modern Schools. In the present experiment no difference was acknowledged between the Grammar and Technical schools.

In the four Grammar and Secondary Modern Schools used in the study all the boys in the classes covering the relevant age range were tested. Similarly all the boys at the Chaigeley School were tested. This avoided possible errors of sampling due to a biased selection of cases.

Administration

The test was administered to groups of boys. It always happened that a group corresponded to an entire class.

The order of presentation was 1M, 2M, 3M, 4M, 5M, 6M, 7M, 8MF, 9MF, 10MF, 11MF.

After the first drawing (1M) had been pinned to the blackboard the following instructions were read out to the class. "I am going to show you pictures like this one at a time. This is a test of your imagination. I want you to make up a story about what you see happening in this picture. As a guide you might like to include what has been happening immediately before the incident shown in the picture, you might also like to include what you believe the people are doing or thinking about or feeling and finally how it all ended. You have got ten minutes in which to finish your story."

At the end of ten minutes the boys were allowed, if they had not already finished, to complete the sentence they were writing. Plate no. 1M was then removed and no. 2M put in its place. The instructions were again repeated as above. This procedure continued until all the plates had been shown, though the instructions were not repeated after the second occasion.

A break of ten to fifteen minutes was always allowed about half-way through the series. At Chaigeley School one class was required to complete the test over two days.

Scoring

Each of the stories was first analysed according to 22 criteria. Each time reference was made to any of these criteria it was scored, no matter how frequently it occurred. The method of scoring was to underline the relevant word, phrase or sentence in the story. Numbers, which accompany the following scoring-criteria, were placed over the section which had been underlined. Examples of "lack of confidence" would carry the figure 19, whilst "poor concentration" would be 7.

The following were the 22 scoring criteria used. Scoring of "significant items" was adopted for actual rather than inferred material.

1. *Daydreaming:*

Evidence that any of the characters in the stories, whether depicted in the drawings or not, liked to daydream, particularly about things that could never come true.

2. *Irritability:*

Evidence that any of the characters in the stories, whether depicted in the drawings or not, was disgruntled, irritable, annoyed or impatient.

3. *Worry:*

Evidence that . . . pondered over their past (either the good or the bad), worried over previous humiliating experiences and difficult situations.

4. *Loneliness:*

Evidence that . . . felt lonely, found difficulty in making new friends or kept in the background on social occasions.

5. *Embarrassment:*

Evidence of embarrassment, blushing, guilt or sensitivity on any subject matter.

6. *Slowness:*

Evidence that any of the characters made up their minds too late.

7. *Concentration:*

Evidence that . . . find difficulty in concentrating on the tasks in hand, or are lost in thought when talking to others, or their minds wander so that they lose track of what they are doing, or that they forget immediately what people have said to them.

8. *Anxiety:*

Evidence of tension, anxiety or nervousness in any of the characters. The following indices would be consistent with this category: mental strain: feeling that difficulties were piling so high that they could not be overcome: a feeling of "going to pieces": losing sleep through worry: nervousness when having to wait: sweating without exercise; and stagefright.

9. *After-thoughts:*

Evidence that any of the characters are bothered by after-thoughts or that they are worrying over something that they should have done but failed to do.

10. *Sleeplessness:*

Evidence of sleeplessness due to worry, excitement, nightmares, or evidence of fitful and disturbed sleep or sleep-walking.

11. *Fatigue:*

Evidence of fatigue and listlessness without apparent reason. This can be accompanied by a feeling of uselessness.

12. *Mood Swings:*

Evidence of extremes of mood—bubbling over with energy or sluggish: happy and depressed without apparent cause: fits of laughing and crying beyond the control of the individual; and frequent ups and downs of mood.

13. *Brooding:*

Evidence that . . . think a great deal about the past, particularly about the good times they had.

14. *Useless Thoughts:*

Evidence that . . . are bothered and worried over something that really does not matter, or that a useless thought comes into their mind repeatedly.

15. *Over-Sensitivity:*

Evidence that . . . are over-sensitive or their feelings are easily hurt, or they take things hard.

16. *Restlessness:*

Evidence that . . . show great restlessness or excitement.

17. *Unhappiness:*

Evidence that . . . are miserable, unhappy or are crying.

18. *Inferiority:*

Evidence that . . . are troubled by feelings of inferiority or self-criticism.

19. *Lack of Confidence:*

Evidence that . . . are lacking in confidence and are shy.

20. *Hypochondriasis:*

Evidence that . . . show concern over their health, think about physical ailments and pains, or are preoccupied with related subjects and professions.

21. *Possible Misfortunes:*

Evidence that . . . are worried over possible misfortune, or are worried excessively over any subject.

22. *Fears:*

Evidence of fears and phobias of any kind.

Statistical Procedures

The procedure was to score the normal and neurotic records on all of the possible signs and to test for the significance between the scores of the two groups on each of these signs. The statistical test which seemed most appropriate was the non-parametric-median test (Siegel, 1956). The median score for the combined group (the median for all scores in the normal and neurotic samples) was calculated, and then both sets of scores were dichotomized at the combined median in a 2 × 2 table. A Chi Square was calculated for the significance of the differences between these frequencies. The χ^2 test used with df=1 was

$$\chi^2 = \frac{N\left((AD-BC)-\frac{N}{2}\right)^2}{(A+B)\,(C+D)\,(A+C)\,(B+D)}$$

χ^2 differences which yielded probabilities of 95 per cent. or higher were used for further analysis. All other scoring criteria were abandoned at this point.

RESULTS

Table 1 shows the mean scores of the normal and maladjusted groups, the χ^2 differences and levels of significance for these differences on the twenty-two scoring criteria. The scores from the two groups proved to be significantly different on nine of the twenty-two scoring criteria.

TABLE I

Mean Scores for the Normal and Maladjusted Groups, the χ^2 Differences and Levels of Significance

Criterion	*Mean Scores* *Normals*	*Maladjusted*	χ^2	*Significance*
1. Daydreaming	·457	·212	1·8	—
2. Irritability	3·338	7·780	24·05	<·001
3. Worry	0	·06	22·0	<·001
4. Loneliness	·277	·17	1·37	—
5. Embarrassment	·628	·723	3·3	<·1>·05
6. Slowness	·012	0	·047	—
7. Concentration	·152	·382	15·04	<·001
8. Anxiety	·155	·170	·027	—
9. After-thought	·018	·021	·51	—
10. Sleeplessness	·003	0	1·1	—
11. Fatigue	·012	·212	36·5	<·001
12. Mood Swings	0	·021	17·2	<·001
13. Brooding	0	·063	29·9	<·001
14. Useless thoughts	0	0	—	—
15. Over-Sensitivity	·027	·148	7·0	<·01>·001
16. Restlessness	·006	·234	63·5	<·001
17. Unhappiness	1·932	1·76	·003	—
18. Inferiority	·158	·297	2·6	—
19. Lack of Confidence	·024	·021	·28	—
20. Hypochondriasis	5·41	5·72	3·06	—
21. Possible Misfortunes	·71	1·042	1·7	—
22. Fears	·152	·276	4·0	<·05>·01

The thirteen criteria which did not differentiate the two groups were abandoned and the records of all the "normal" and maladjusted children scored on the basis of the nine remaining criteria. Table 2 shows the percentage frequencies for the different scores as calculated for the two groups.

TABLE II

Percentage Distribution of Scores for the Normal and Maladjusted Groups

Score	*Normals* Percentage at or below score (to nearest whole number).	*Maladjusted* Percentage at or above score (to nearest whole number).
23	100	
22	100	
21	100	
20	100	0
19	100	6

Table II—continued

Score	*Normals*	*Maladjusted*
18	100	13
17	99	19
16	99	21
15	99	21
14	98	21
13	98	21
12	97	26
11	97	32
10	96	38
9	95	47
8	94	53
7	90	66
6	85	77
5	79	79
4	73	85
3	60	91
2	40	96
1	26	100
0	12	100

The differentiation afforded by the revised scoring scheme appears to be of diagnostic value.* The optimum cut-off point appears to be five. Seventy-nine per cent. of the maladjusted boys scored at or above this point, whilst seventy-nine per cent. of the "normal" boys scored at or below this point. As the scores go beyond five, the number of normals scoring at these points decrease rapidly. For example, ninety-four per cent. of the normal boys scored at or below eight, whilst as many as fifty-three per cent. of the maladjusted boys scored at or above that point.

DISCUSSION AND CONCLUSIONS

The suggested approach of combining the projective and questionnaire methods thus leads to results of apparent diagnostic significance. Confidence in the revised scoring scheme must however await further cross-validatory studies.

The results obtained from the records of the 328 "normal" boys are interesting for several other reasons.

Firstly, on the majority of the twenty-two criteria the mean scores were very small, though on the "irritability", "unhappiness" and "hypochondriasis" criteria the scores were high.

Certain tentative suggestions might be made to explain these results. It will be remembered that the "irritability" criterion was scored when there was evidence that any of the characters in the stories were disgruntled, irritable, annoyed or impatient. In the stories the characters were often said to become bored because they did not know what to do; parents punished children for wrongdoing; teachers made the boys stay in after school to do extra work; boys received a "good hiding" from the parents; boys fought for supremacy; boys were annoyed because they had lots of homework to do and they wanted to play outside; boys were put into detention at school; creation of adventurous themes of murder and acts of violence; pocket-money stopped by the parents because the boy had broken a window and was forced to pay for it; and evidence of jealousy. These themes occurred time and time again. They suggest that boys in the present age range might be very much concerned with the problem of socialization and the restrictions imposed by parents, teachers and police. They frequently create stories in which they find the process of socialization and its restrictions running contrary to their wishes.

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* Copies of the photographic plates, lists of significant scoring responses and the distribution of scores of the normal sample on the 22 scoring criteria, can be obtained on application.

A further aspect of this problem is the greater frequency with which themes of "irritability" occur in the maladjusted group. The intensity of the responses is also greater. Several examples from the maladjusted group will emphasize this -"Slash—a person slashes another with a razor"—"she said she was going to kill it (baby)"—"he was pushed down the stairs"—"his father was waiting for him with a snarl on his face"—"shall I set fire to the place, because I have nothing to do?"

"Unhappiness" was one of the most frequent responses in the normal sample. The fact that depression is a common feature of adult neurosis whilst no significant differences were demonstrated between the present two groups is interesting in itself.

The criterion scored most frequently in the normal group was "hypochondriasis". Boys in the present age range readily create stories in which injury, doctors, hospitals and illness predominate.

Several of the scoring criteria which successfully differentiated the two groups seem to hang together. These criteria suggest that the maladjusted child is unsettled, easily irritated, not able to concentrate and restless.

Symonds' work (1949) is relevant to the present study as his results appear to validate the present findings. Forty-two stories were collected from each of forty normal adolescent boys and girls in response to a thematic apperception test. The 1,680 stories were analysed for their thematic content and classified. His work is important as it represents one of the few pieces of experimental work with projective techniques in which normal data have been collected. Twenty-seven of the forty subjects were of a similar age to the present sample. His work represents an opportunity to compare types of response from two independent normal samples and to decide on the relative reliability of the present findings. Both Symonds' work and the present study, combined, represent an analysis of 5,805 stories.

His first finding is of particular interest. He noted that every adolescent boy and girl told at least three stories including themes of aggression. These ranged from mild criticism and rebuke to robbery and murder. Stories were frequently found to contain references to destruction and violence, often of a criminal nature. Frequently also aggression was caused by rivalry and jealousy. Punishment was again a frequent theme particularly with regard to school. Teachers were almost always stern, threatening and avenging. This emphasis on aggressive content is important as the same trend was noted in the present sample under "irritability".

A further finding of importance by Symonds was that happiness occurred less frequently as a theme than depression. In the present study "unhappiness" was a frequent response.

Two further themes occurred frequently in Symonds' work. The first involved conflict of good with evil so that guilt and conscience appeared in many of the stories. The second consisted of themes reflecting a fear of sickness, accident and injury. These themes ranked fourth and first in frequency in the present normal sample.

Comparing the two studies there is an encouraging measure of agreement between them on the relative emphasis of aggressive, depressive, guilt and hypochondriacal themes in normals. In one way Symonds' work has further validated the present results.

A more recent report (Lyle and Gilchrist, 1958) provided further confirmatory evidence of the validity of the present results. Two groups of boys were matched for age. One group consisted of 31 delinquent boys, all of whom had recently been charged with offences at a children's court. The other group consisted of 31 non-delinquents who attended a technical school in a working-class area which was officially recognized as a "delinquency area" in Melbourne. Very few of the boys in the two samples were considered to be maladjusted, though one was considered to be potentially psychotic. Six T.A.T. cards were administered to each member of the two groups. The experiment represented, therefore, an opportunity to test further a prediction arising out of the present results. Since Lyle and Gilchrist's samples contained few maladjusted boys, then the two groups should not be differentiated on the basis of "irritability", in spite of assertiveness and destructiveness (Glueck and Glueck, 1950), extrapunitive (Grygier, 1954) and antisocial behaviour (Barry *et al.*, 1956) having been found frequently in groups of delinquent males. The T.A.T. stories of the 31 delinquent and 31 unselected non-delinquents were classified according to their themes using the six cards. No significant differences emerged between delinquents and non-delinquents for the aggressive, antisocial, percentory or punishment themes. Lyle and Gilchrist conclude, "This type of analysis therefore fails to support the hypothesis, that delinquents would give more aggressive, antisocial themes than non-delinquents in their T.A.T. protocols". The conclusion arising from their study, that of Symonds and the present work is that "irritability" appears to be a theme common to the stories of normal and delinquent boys, though it occurs more frequently in the stories of maladjusted boys. The present findings have thus been further supported in that a prediction based on test responses has been confirmed in spite of contrary evidence having been suggested by behavioural characteristics commonly observed in delinquents.

Several conclusions are suggested by the present work:

1. The fact that significant differences have been found between the scores of the two groups suggests the general validity of the present approach. This was to the effect that verifiable predictions can be made from the adult experimental field into the field of personality assessment in children.

2. The data available from the present normal sample suggests that large scores may occur on certain of the scoring criteria. In the absence of normal data suggesting such trends boys might easily have been falsely diagnosed, for example, as "over preoccupied with health" or "over irritable and aggressive".

3. The present investigation has provided a tentative confirmation of Symonds' work. Both sets of data together suggest that maladjustment might involve, not distinctions of quality, but only differences of degree.

SUMMARY

The present experiment was designed to check the suggestion that a combination of the projective and questionnaire approaches might be of value in developing a valid test of neuroticism for use with children.

A new series of projective plates, related to an English culture pattern, was constructed. A new method of scoring was also devised based on four questionnaires of adult neuroticism.

The test was administered to 328 "normal" boys between the ages of 11 and 14 and to 47 maladjusted boys aged between 11 and 16. The scoring criteria which failed to distinguish the maladjusted from the normal boys were abandoned. The records of

the two groups of boys were then assessed on the basis of the nine remaining scoring criteria. The results were encouraging and suggest that the test may be of diagnostic value. Using a cut-off point of five, seventy-nine per cent. of the maladjusted boys scored at or higher than this point, whilst seventy-nine per cent. of the normal boys scored at or lower than the cut-off point.

The results obtained by Symonds and Lyle and Gilchrist, both using normal children, were confirmed. The present results suggest that childhood maladjustment differs from normality quantitatively rather than qualitatively.

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PITUITARY SNUFF, PROPANTHELINE AND PLACEBOS IN THE TREATMENT OF ENURESIS

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CONTROL of micturition by day should ordinarily be acquired in the second year of life and by night in the third, and enuresis comprises repeated involuntary micturition after this age if not due to organic disease. If the latter is responsible the symptom is termed incontinence of urine (Hubble, 1950).

The variety of theories of aetiology is rivalled only by that of attempts at therapy but it is not our intention in this paper to present a comprehensive survey of the literature nor to stress any single viewpoint as to causation. The aims of the present study were in fact:

1. To conduct a controlled therapeutic trial of posterior pituitary snuff ("Di-sipidin") and propantheline bromide ("Pro-Banthine").
2. To discuss as a problem in its own right, the role of the placebos used.
3. To relate responses under (1) and (2) to the factors of age, degree of neuroticism and previous remission if such occurred.

Physiopathology

The bladder receives its sympathetic nerve supply from spinal cord segments L.1 and L.2 and its sacral autonomic supply largely from S2. The internal sphincter is contracted by sympathetic and relaxed by sacral autonomic stimulation. The latter also produces stimulation of the detrusor while it is probable that the sympathetic can exert an inhibitory effect on the body of the bladder. Afferent fibres probably follow a dual route (Wright, 1952). Local innervation is under cortical influence, the exact role of which seems at present not fully defined.

Various aspects of the neurological system have been emphasized in theories of causation of enuresis. Thus Stalker and Band (1946) have discussed the finding of an uninhibited neurogenic pattern on cystometrography, and the possibility of enuresis being a local autonomic syndrome released by lack of cerebral inhibition; Ström-Olsen (1950) has suggested that the defect might lie in some inborn error in cortical neurone physiology resulting in functional interruption of cortical pathways to the hypothalamus producing the syndrome of deep sleep and loss of bladder control; and Jennings Marshall (1954) has written of the possibility of parasympatheticotonia or of ectopic distribution of innervation to the prostatic urethra.

Bakwin (1938) suggested an anatomical basis in innervation for his concept of the irritable bladder and Braithwaite (1955) who made extensive cystometric studies, postulated detrusor achalasia, but Poulton and Hinden (1953) classified only seven of two hundred enuretic children under the latter heading. In the last quoted study, however, various types of polyuria took numerical pride of place, 154 patients being found to show absolute polyuria or relative nocturnal polyuria or a combination of these, and their volumetric studies thus stressed the first factor in the relationship between urine volume and bladder tolerance, rather than purely neurological considerations. The significance of polyuria and whether it is a primary or secondary phenomenon, is somewhat obscure: for example it might be due to emotional disturbance caused by, or possibly causing, enuresis while a further possibility lies in a primary bladder neck abnormality (*Lancet*, 1955).

Psychopathology

In the "normal" child as Holman (1954) points out, physical development makes bladder control possible while mental development makes it desired. A brief survey of the literature of enuresis discloses discrepancies in the assessment of the frequency of psychological causation and no doubt selection of patients and the orientation of the doctor go far to account for this.

Bakwin (1938) expressed the view that psychic factors had an important secondary role but were of primary influence only in a few cases and Poulton and Hinden (1953) estimated that only twelve of their two hundred cases could be regarded as psychogenic wetting. On the other hand Gerard (1939) attributed organic causation to only seven of her series of seventy-two enuretic patients and faulty training to four more and Strauss (1944) considered that by far the greater number of cases of enuresis were of psychological rather than urological importance. Stalker and Band (1946) provided a psychiatric diagnosis for all but sixteen of their sixty-seven post-pubertal patients and these sixteen were classified as anxious personalities although not of abnormal degree.

Kanner (1948) remarked that enuresis is almost always combined with other expressions of emotional maladjustment, a view supported by the work of Michaels and Goodman (1934) who found an abnormally raised incidence of certain "neuropathic traits" associated with enuresis.

Psychoanalytically (Fenichel, 1946) active wetting has been described as originally giving auto-erotic pleasure while later enuresis may develop as an involuntary nervous symptom, an unconscious equivalent of masturbation. Gerard (1939) in the series quoted above found that sixty-one of her patients presented definite neurotic patterns and that in the largest sub-group enuresis was part of a syndrome with a common aetiology based on fear of harm from persons of the opposite sex.

At a clinical level such factors as the birth of a sibling or a frictional parental relationship are often of crucial importance and this is also true of emotional attitudes precipitated in child and parent by the bed wetting itself. Such attitudes may well involve guilt and resentment and, as Kanner (1948) observes, amalgamation of primary and secondary phenomena is noteworthy so that improvement in the latter touches on some of the fundamental conflicts. While an emotional change may be inevitable if the symptom is to be resolved, it is not always necessary wittingly to invoke deep unconscious mechanisms and a fairly superficial psychotherapeutic approach may be highly successful. Drugs too may have a psychodynamic effect, quite apart from any pharmacological action, and this is given additional emphasis by reports such as that of Friedell (1927) who claimed a cure rate of 87 per cent. by a regime which included the injection of sterile water combined, it is true, with fluid restriction.

Rigid separation of psychological from physical concepts is unjustifiable and lack of cortical inhibition or local autonomic dysfunction may merely represent the somatic matrix and means of expression of psychic influences. It is, however, the authors' experience that enuresis may occur in the absence, not only of demonstrable physical disease, but also of convincing psychiatric disorder. Thus in the present series of 44 physically fit enuretic individuals 7 (15·9 per cent.) would have been regarded as psychiatrically normal too but for the occurrence of enuresis.

METHOD OF THE PRESENT INVESTIGATION

In comparison with many psychiatric studies enuresis lends itself to statistical evaluation. Nevertheless there is the factor of human fallibility in recording, though this should be balanced by the use of controls, and in addition it is not practicable to measure degrees of wetness. In this study a night was graded as wet if there were any doubt about it.

Forty-four patients were studied in the present series and in all cases organic causation had been excluded by investigation of varying complexity. There was no formal selection of patients once they were referred to the psychiatric clinic, other than the exclusion, with four exceptions, of those under eight years of age, and of a very few on the grounds of inability of the parents to co-operate fully or where there was a rapidly changing environmental situation. It was also required that patients should have had no previous experience of the drugs used.

Essentially the same procedure was adopted for both the pituitary snuff and the propantheline trials. The patient and/or parent were informed that a new treatment was being tried and requested (1) to keep a record of dry and wet nights, (2) to avoid fluids for two hours before retiring, (3) not to vary the normal daily routine more than could be helped but to make a note of any special or unusual events, and (4) not to employ any other treatment than that prescribed. The co-operation of the family doctors was sought especially with regard to not giving any additional drugs during the period of the trial. Monthly review at the psychiatric departments was arranged and on these occasions psychotherapy was restricted to simple reassurance.

At the first attendance a record sheet was completed which incorporated in itemized form a full history together with a clinical estimate of intelligence and of various personality traits and a note as to the occurrence of previous remissions if any.

The patients were graded according to the following criteria:

Grade I Apart from the enuresis the patient and his home and school or work situation appeared essentially normal.

Grade II There was a "nervous" or over-sensitive or anxious disposition but apart from the enuresis the patients might not have been considered sufficiently abnormal to justify psychiatric attention.

Grade III There was strong actual or presumptive evidence of psychiatric disturbance, for example in the form of night terrors, habit spasms or poor adjustment to school in children or overt neurotic symptoms, such as pathological anxiety, in adults.

THE POSTERIOR PITUITARY SNUFF TRIAL

The role of the posterior pituitary in the control of water excretion and osmotic equilibrium is familiar from the classical studies of Verney (1957) and the antidiuretic effect of appropriate extracts is exploited in the treatment of diabetes insipidus but is evident in normal individuals too. The use of such extracts in nocturnal enuresis could perhaps most properly be confined to those patients in the polyuric group but by altering the ratio urine volume : bladder tolerance other enuretic patterns might be expected to benefit. Previous controlled therapeutic studies include those of Marson (1955, 1956), who reported considerable success in six adolescent and adult patients, and Holt (1956), who obtained less spectacular results in children.

Thirty-four patients took part in the present trial, their age and sex distribution being shown in Table I.

TABLE I

Age	<8	8	9	10	11	12	13	14	15	16	18	19	22	Total
Male ..	.. 1	3	5	3	2	2	3	1	–	1	1	1	–	23
Female	.. –	4	–	1	2	–	–	1	1	–	–	1	1	11
Total	.. 1	7	5	4	4	2	3	2	1	1	1	2	1	34

Prescriptions were issued at monthly intervals and at each attendance the record kept by the patient or parent was summarized as percentage of dry and wet nights. The active snuff ("Di-sipidin"), obtained from the posterior lobes of beef pituitary glands and with an added lactose diluent, was administered in capsular form using the Di-sipidin insufflator, each capsule having 30 A.D units potency. The placebo was administered in identical form but was composed of powdered lymphatic gland, hog stomach and lactose in the proportions of 5:1:4. Doses of one capsule at bedtime were employed and only the pharmacist, until the end of the whole trial, was aware of whether active or placebo preparation was being used.

The results of the trial were assessed by calculating for each patient the proportions of dry nights over the trial referable to the periods when posterior pituitary snuff and placebo respectively were being administered, and the proportion of dry nights for the whole trial was also obtained. The degree of improvement is defined as the difference between such a proportion and the frequency of dry nights before treatment commenced and an arbitrary four-point scale is used. (Most patients were wet every night before starting treatment.) In addition to this type of comparison the actual proportions of dry nights for various groups of patients whilst on posterior pituitary snuff and placebo respectively are given and distinction is made between weighted proportions which take account of the actual totals of dry nights of a group of patients and unweighted proportions, viz. the means of the individual percentages of dry nights of patients in the group.

Table II shows that over the period of the trial, 15 patients (44·1 per cent) achieved proportions of dry nights which exceeded their proportions at the beginning of the trial by 30 per cent. or more. Over one-third of the patients had a proportion of dry nights on posterior pituitary snuff which exceeded the placebo proportion by 10 per cent. or more; and of the 15 patients with moderate or good improvement 7 had a more favourable response at this level of difference whilst on the active preparation.

TABLE II

Degree of Improvement	Total No. of Cases	Active Snuff Proportion Exceeds Placebo by 10 per cent. or More	Less than 10 per cent. Difference Between the Proportions	Placebo Proportion Exceeds Active Snuff by 10 per cent. or More
Negligible (<10 per cent.)	12 (35·3)	2	10	–
Slight (10–29 per cent.) ..	7 (20·6)	3	3	1
Moderate (30–59 per cent.)	14 (41·2)	7	7	–
Good (60 per cent. and over)	1 (2·9)	—	1	–
Total	34 (100·0)	12	21	1

Improvements achieved whilst on posterior pituitary snuff and placebo separately are given in Table III and patients have also been grouped according to age. For each age group the mean proportion of dry nights for the active preparation exceeded the corresponding placebo proportion. Comparison of the paired distributions shows that only one or two patients in each age group fell into a higher improvement group while on the active substance, which confirms the above finding. It is also clear that patients aged 15 years and over responded better than did those in the younger age groups, and the gradient of increasing improvement with increasing age is only interrupted at the 13–14 year level.

TABLE III

Degree of Improvement	Age Groups <9		9–		11–		13–		15 and Over		Total for All Ages	
	A	P	A	P	A	P	A	P	A	P	A	P
Negligible (<10%) ..	3	3	4	5	2	2	3	3	–	1	12	14
Slight (10% to 29%) ..	3	2	3	3	–	2	–	1	2	1	8	9
Moderate (30% to 59%)	–	2	2	1	3	2	2	1	4	4	11	10
Good (60% and over) ..	2	1	–	–	1	–	–	–	–	–	3	1
Total	8	8	9	9	6	6	5	5	6	6	34	34
Mean proportion of dry nights (per cent.):												
Weighted	35·8	28·7	38·2	31·9	66·5	54·5	49·0	40·0	86·8	71·6	51·7	42·1
Unweighted ..	33·9	29·4	31·7	28·8	66·4	55·3	49·9	41·7	86·6	74·5	50·7	43·6

In this and subsequent Tables: A=potentially active preparation and P=placebo.

The effect of grading on response was examined and Table IV gives the improvement distributions and mean proportions of dry nights for each grade.

TABLE IV

Degree of Improvement	Grade I		II		III	
	A	P	A	P	A	P
Negligible (<10%) ..	3	3	5	7	4	4
Slight (10% to 29%) ..	–	–	3	5	5	4
Moderate (30% to 59%)	1	2	8	5	2	3
Good (60% and over) ..	1	–	2	1	–	–
Total	5	5	18	18	11	11
Mean proportion of:						
Weighted	55·7%	41·7%	56·6%	45·9%	41·2%	36·5%
Unweighted	55·0%	44·2%	55·4%	46·7%	41·0%	38·1%

The posterior pituitary snuff proportion (weighted and unweighted) exceeded the placebo for each grade and results for Grade III were less satisfactory than those for Grade II. Only 5 patients were in Grade I but their results agree closely with those for Grade II.

Patients were next divided into those with previous remissions of one month or more and those with remissions of less than this or none at all. As can be seen from Table V, patients in the former group responded more favourably and this was true of both posterior pituitary snuff and placebo.

TABLE V

Degree of Improvement	Length of Previous Remission: None or Less than One Month		One Month or Longer	
	A	P	A	P
Negligible (<10%)	8	9	4	5
Slight (10% to 29%)	6	7	2	2
Moderate (30% to 59%)	6	6	5	4
Good (60%)	2	–	1	1
Total	22	22	12	12
Mean proportion of dry nights:				
Weighted	44·8%	35·2%	63·9%	56·7%
Unweighted	43·2%	35·6%	64·4%	58·2%

It might be expected that older patients would be more likely to have a history of remission, but separate analysis of patients over 13 years old showed that previous remission still favoured improvement. For each group the active snuff mean proportion of dry nights exceeded the placebo value.

It is evident from these results that almost half the patients improved to the extent of an increase in the proportion of dry nights of 30 per cent. or more and over one-third of the patients had a proportion of dry nights while on posterior pituitary snuff which exceeded the placebo proportion by 10 per cent. or more. For each of the factors of age, grade and previous remission the results were better with the active preparation than with the placebo.

THE PROPANTHELINE TRIAL

Methanthelinium bromide ("Banthine") and propantheline bromide ("Pro-Banthine") are anti-cholinergic compounds, the latter being more potent and with less troublesome side effects (*Extra Pharmacopoeia*, 1958). The action of the former on normal and various types of neurogenic bladders has been studied cystometrically by Keizur and Hodges (1953) who concluded that it exerted a profound depressant effect on the detrusor muscle via partial block of parasympathetic innervation.

Propantheline has been used in controlled trials of the treatment of enuresis by Leys (1956), Mayon-White (1956), and Holt (1956), with results ranging from limited but definite to no observable effect. Braithwaite (1955) without using controls, reported 7 out of 33 cured or greatly improved but obtained his best results of all by combination with amphetamine, 10 out of 13 being cured in this way. If effective at all propantheline might be expected to benefit most those cases showing the irritable bladder syndrome, but as with posterior pituitary snuff, it might so alter the relationship between urine volume and bladder tolerance as to be helpful in other forms of enuresis.

Twenty-five patients took part in the trial and their age and sex distribution are shown in Table VI. Fifteen of these patients were included in the preceding trial with posterior pituitary snuff.

TABLE VI

Age	<8	8–	9–	10–	11–	12–	13–	14–	Total
Male	3	2	3	1	2	2	3	2	18
Female ..	1	3	1	–	1	–	–	1	7
Total ..	4	5	4	1	3	2	3	3	25

Prescriptions were again issued at monthly intervals but units of a fortnight's duration were chosen for comparison and only the pharmacist, until the end of the trial, knew when active preparation or placebo was supplied. The dose of propantheline was 30–45 mg. to be taken at bed-time, and the placebo tablet (composed of a sugar core and coating) was of identical appearance. At each attendance the usual note of dry and wet nights was made for each fortnight separately and any unusual occurrences were recorded.

The methods of assessment of results were as described for the posterior pituitary snuff trial. For those patients who were also included in the latter trial the proportion of dry nights during the last month of it was taken as the base line from which the degree of improvement was measured.

Table VII shows that one-fifth of the patients have proportions of dry nights over the whole trial which exceeded their initial proportions by at least 30 per cent. Almost one-third had a proportion of dry nights while on propantheline which exceeded the placebo proportion by 10 per cent. or more. However, none of the eight patients concerned showed moderate or good improvement, and it will be noted that of the five patients who improved by 30 per cent. or more two had placebo proportions which exceeded the corresponding propantheline proportions by 10 per cent. or more.

TABLE VII

Degree of Improvement	Total No. of Cases	Number of Cases in which: Propantheline Proportion Exceeds Placebo by 10% or More	Less than 10% Difference Between the Proportions	Placebo Proportion Exceeds Propantheline by 10% or More
Negligible (<10%) ..	12 (48·0%)	4	8	–
Slight (10% to 29%) ..	8 (32·0%)	4	3	1
Moderate (30% to 59%)	4 (16·0%)	–	2	2
Good (60% and over) ..	1 (4·0%)	–	1	–
Total	25 (100·0%)	8	14	3

Comparison of the degree of improvement achieved while on propantheline and placebo respectively is shown in Table VIII which gives distributions for patients according to age. It is evident that seven patients showed slight improvement while on propantheline but negligible improvement while the inert

TABLE VIII

Degree of Improvement	Age Groups 9		9–11		12 and Over		Total for All Ages	
	A	P	A	P	A	P	A	P
Negligible (<10%)	4	7	3	5	2	4	9	16
Slight (10% to 29%)	4	2	3	1	4	–	11	3
Moderate (30% to 59%) ..	1	–	2	2	1	3	4	5
Good (60% and over) ..	–	–	–	–	1	1	1	1
Total	9	9	8	8	8	8	25	25
Mean proportion of dry nights (per cent.):								
Weighted	31·3	22·2	58·4	51·3	57·2	56·8	45·9	41·6
Unweighted	29·5	13·1	57·1	48·6	58·0	57·7	47·5	41·1

substance was administered. Such differences between the two distributions are in accord with the small difference between the mean proportions of dry nights for the two substances when all patients are considered. From the paired distributions and mean proportions it is evident that patients under nine years of age did not respond so well as those in the two older age-groups between which however there was little difference. Propantheline was more efficacious than placebo only for patients under nine years old.

The effect of grading upon response to propantheline is shown in Table IX.

TABLE IX

Degree of Improvement	Grade I		II		III	
	A	P	A	P	A	P
Negligible (<10%) ..	3	3	3	6	3	7
Slight (10% to 29%) ..	2	–	3	–	6	3
Moderate (30% to 59%)	–	2	2	2	2	1
Good (60% and over) ..	–	–	1	1	–	–
Total	5	5	9	9	11	11
Mean proportion of dry nights:						
Weighted	33·6%	46·5%	61·7%	56·9%	39·6%	29·2%
Unweighted ..	35·6%	42·5%	62·1%	57·2%	40·9%	27·4%

Results for Grade II patients were better than those for either Grade I or Grade III patients and the placebo was superior to propantheline for Grade I patients. The number of the latter however was very small and the difference was barely significant.

Finally the improvement distributions are given in Table X for patients classified according to whether or not they had previously had a remission of

TABLE X

Degree of Improvement	Length of Previous Remission None or Less than One Month		One Month or Longer	
	A	P	A	P
Negligible (<10%)	5	11	4	5
Slight (10% to 29%)	9	3	2	–
Moderate (30% to 59%)	4	4	–	1
Good (60% and over)	–	–	1	1
Total	18	18	7	7
Mean proportion of dry nights:				
Weighted	39·0%	35·0%	65·3%	62·2%
Unweighted	41·2%	33·4%	63·6%	61·1%

one month or more. Again, and in each group, some patients obtained slight improvement while on propantheline but negligible improvement while on placebo; there was little difference between mean values. A history of remission was, as in the posterior pituitary snuff trial, associated with better results.

DISCUSSION

We should like to anticipate some of the possible criticisms of the trial before attempting to draw any conclusions from it. Although the numbers in this trial compare favourably with some studies, they are nevertheless smaller than we should have liked and this is particularly true where the results are broken down into subgroups. These latter results have been considered significant when they show consistent trends within any one trial and especially between the two trials.

The figures for incidence of wet nights before the treatment, especially in the pituitary snuff series, depended on recollections rather than day to day recordings and cannot be regarded as completely accurate. At the same time we were much impressed by the scrupulous co-operation of parents and patients and, where this was not apparent, the cases were not included.

The placebo in the pituitary snuff trial cannot be regarded as wholly inert, but there is no reason to believe that the substances involved would influence enuresis by intrinsic action. A few patients were able to detect the difference between the placebo and the pituitary snuff preparation but had no reason for greater confidence in one than the other.

It is an inescapable conclusion that the main response in both trials is due to placebo reaction, and there is good consistency between the placebo responses in the two trials (see "total for all ages" column in Tables III and VIII). Since many of the patients had already received various treatments (but not those prescribed by us) for their enuresis before referral and since deliberate suggestion was avoided, the basic figure for placebo response is a low estimate and could probably be increased by deliberate suggestion in patients receiving treatment for the first time. We had hoped to examine placebo reaction in relation to several factors but it became apparent that the response in young patients was complex, involving a reaction both in parents and child. For this reason we have confined ourselves to three fairly simple considerations namely age, degree of neurotic disturbance and previous remission.

Since the taking of snuff is a more novel and impressive procedure than swallowing a tablet, we had expected that there would be a higher placebo response in the pituitary snuff trial but this was not the case. Another unexpected finding was that those patients, who had responded poorly to snuff, did rather better on changing to propantheline than did the other patients in the propantheline trial.

Separate analyses were made of males and females but these showed no marked differences in the response distributions, although relatively few female patients entered the trial.

The most favourable factor for a positive response is a previous remission of a month or over (Tables V and X). In general, age favours a positive response (Tables III and VIII) and the ages of 11 for the pituitary snuff trial and 9 for the propantheline trial appear to have some critical significance. In the pituitary snuff trial the relatively low figures for the 13–14 group (Table III) are alone inconsistent and may be accounted for by small numbers. Many doctors regard the adult enuretic as a particularly difficult patient to treat but this does not find support, at any rate where unco-operative patients are excluded.

Grade III patients showed less response than Grades I and II (Tables IV and IX) except with the active preparation in the propantheline series where the results were worst in Grade I but the number concerned is small. In the same series Grade II did better than Grade I but this trend was not significantly present with the posterior pituitary snuff series. These observations are in accord with the following speculation. Grade III is made up of patients with poor personality structure, unfavourable environment and multiple neurotic symptoms and the total situation and use of symptoms for secondary gain, in many cases, could be too overwhelming to permit a positive placebo response. In Grade II there is some tension and neurotic drive but a favourable environment and little secondary gain, factors which are probably ideal for a placebo response. In Grade I neuroticism and abnormal environments were not detected so that there might be less potential for response to suggestion. Such a view is in keeping with that advanced by Tibbetts and Hawkings (1956) who made studies of placebo response in adults. They found a better placebo response in a trial where case selection was more discriminating compared with another trial which included patients with poorer personality structure and greater evidence of hysterical conversion (Hawkings and Tibbetts, 1956a, 1956b). They conclude "This trend is to the effect that patients with unelaborated anxiety are those most likely to respond in a positive manner to a placebo administered under the circumstances described. Somatic elaboration with a physiological basis does not greatly prejudice this response, but hysterical conversion affects it adversely. The fuller the conversion, the more resistant is the patient."

The response in subgroups follows a remarkably similar pattern where active and control substances are compared throughout both trials, thus confirming the suspicion that suggestion is playing a major part where pituitary snuff and propantheline are prescribed. Even so there is significant evidence that the active substances are more effective, especially where pituitary snuff is concerned. We suggest that this is due to pharmacological action in a marginal group where the lessened urinary secretion (posterior pituitary snuff) or increased bladder tolerance (propantheline) are critical for dryness or wetness. Many parents observed independently in the course of the trial that the degree of wetness was much reduced but we thought this was too nebulous to take into account and felt bound to take dryness or wetness as the only criteria. Nevertheless the drugs may be more potent in the treatment of enuresis than the trial indicates.

In this experiment we have been forced to standardize treatment so far as possible, but in order to obtain the best results in the treatment of enuretics, "total push" is probably the best procedure. This might involve a combination of drugs such as posterior pituitary snuff, propantheline and amphetamine. Children could be woken during the night and given a further dose to ensure action throughout the whole period of sleep. Certainly support and encouragement for the parents are nearly always indicated and a few specially selected children should receive psychotherapy rather than treatment by drugs of any kind.

SUMMARY

Controlled trials of posterior pituitary snuff and of propantheline were conducted on a series of forty-four enuretic patients.

Responses to the active preparation and to the placebos employed were related to the factors of age, grade of neuroticism and previous remission.

It was concluded that the main response in both trials was due to placebo reaction but there was a marginal area where the active preparations, especially posterior pituitary snuff, were pharmacologically effective.

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THE INFLUENCE OF AMOBARBITAL (AMYLOBARBITONE) AND METHAMPHETAMINE ON THE FOCUS OF ATTENTION*

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INTRODUCTION

CERTAIN drugs apparently alter the focus of attention. This paper deals with attempts to confirm and extend knowledge about drug influences on the focus of attention, and to relate the concept of the focus of attention to the concepts of extraversion and introversion used by Eysenck and his co-workers.

Attention may be conceived of as two complementary processes. On one hand, there is a "process of selecting one out of several simultaneously possible objects or trains of thought" (16). On the other hand, there is a process of "withdrawing from" or ignoring peripheral factors. The focus of attention determines the degree to which peripheral factors are ignored. If factors lying outside the central focus of attention easily influence behaviour, we may infer that the process of ignoring these factors is minimal, or that the focus of attention is broad. If peripheral factors are being effectively ignored, and so have little or no effect on behaviour, then we may infer that the focus of attention is narrowed.

The focus of attention can be artificially altered by a number of different methods. For example, the focus of attention may be narrowed by inhalations of amyl nitrite, by a cold pressor procedure, by an anti-cholinesterase nerve gas, and by methamphetamine. This is a rather heterogeneous list since amyl nitrite is a vasodilator; the cold pressor procedure is vasoconstricting; methamphetamine is a sympathomimetic; and anti-cholinesterases are parasympathomimetic. All of these procedures have one thing in common; they all produce a more "alert" EEG. Nerve gas seems capable of producing an alert EEG without causing behavioural arousal; so, since atropine can produce a drowsy EEG without producing behavioural drowsiness, atropine was tried as a likely attention broadening agent. The predicted effect was found following two milligrams of atropine sulphate injected intramuscularly (5). These data suggest a correlation between the focus of attention and some neurophysiological phenomena underlying EEG arousal. Neurophysiological evidence supporting the reasonableness of such an assumption has been presented elsewhere (4, 5, 6).

A number of ways to measure focus of attention were developed for use in the various attention-narrowing experiments, but none of these measures were

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suitable for use with atropine. Consequently new techniques had to be introduced for studying atropine as a potential attention broadener. Out of several procedures tried, a combined score derived from the Stroop test and the Embedded Figures test was most sensitive. These two tests will be described in detail under *Experimental Procedures*. After the atropine experiments, however, it still remained to be seen whether these apparent indicators of broadened attention would also be sensitive to narrowed attention. To accomplish this purpose we selected methamphetamine as the most reliable attention narrower and set out to determine the influence of methamphetamine on Stroop and Embedded Figures test performance. The guessing game had previously been used in the study of methamphetamine. However the results were reliable only at the 5 per cent. level of confidence by one-tailed test. Because of this it seemed advisable to repeat the observations on the effects of methamphetamine on guessing game performance.

Finally, from a purely theoretical standpoint, the concept of focus of attention and the continuum of extraversion-introversion would seem to have some things in common. As Evans and McConnell (9) put it: ". . . the thinking introvert . . . tends not to be so dominated or oriented by objective conditions . . ." In addition to this, Eysenck and his co-workers (10, 11, 12, 13, 14) found dexedrine to have "introverting effects". Dexedrine is pharmacologically similar to methamphetamine, and methamphetamine is found to narrow attention. Finally, they reported that amobarbital (amylobarbitone) in doses of 4·5 grains has "extraverting effects". Because of amobarbital's effect on neurophysiological arousal, amobarbital would be expected to broaden attention. With these thoughts in mind, amobarbital was included as a drug in the main set of experiments to be reported. The Maudsley Personality Inventory was also used to measure both neuroticism and extraversion so that intercorrelations between these measures and measures of the focus of attention could be determined. In a preliminary experiment, low correlations were found between introversion and narrowing of attention and also between neuroticism and narrowing of attention. These correlations were not significant but seemed suggestive enough to justify testing the possibility of such relationships on a larger sample.

EXPERIMENT

(*a*) SUBJECTS

The subjects for these experiments were 28 Johns Hopkins University undergraduate students recruited by the offer of four dollars for about two hours time. Subjects were excluded if they were known to be taking centrally active drugs or were in a distinctly abnormal condition at the time of testing. Thus, the testing of two subjects was postponed because of severe hangovers. Usual tobacco and coffee consumption was permitted.

(*b*) METHOD

On the day of testing the subject was brought into the laboratory, allowed to read a simplified protocol of the experimental procedure, and asked to sign a release. He was then put through the testing procedure outlined in Table I.

Subjects were assigned to three different drug treatments so as to maintain a balanced distribution of initial Stroop scores. No attempt was made to disguise the nature of the medication from the technician who had administered the test because of practical considerations. However, post-test interviewing of the subjects revealed that none of them were aware of the nature of their medication

TABLE I

Schedule for Experiment

A. Pre-Drug Schedule:
   1. Instructions and sign release.
   2. Maudsley Personality Inventory.
   3. Progressive matrices.
   4. Stroop colour-word interference test (Stroop 1).

B. Drug—one of the three, immediately following Stroop 1:
   1. Placebo (2 tablets of milk sugar).
   2. Methamphetamine (2 tablets, 5 mg. each).
   3. Na Amobarbital (1 cap. 65 mg. and 1 cap. 200 mg. Total: 265 mg. (approx. 4·9 grains)). (To make the closest approach to the 4·5 grain dosage used by Eysenck and his group.)

C. Post-Drug Tests (begun exactly 1 hour after end of Stroop Test 1):
   1. Repeat Stroop colour-word interference test (Stroop 2).
   2. Witkin's coloured Embedded Figures Test.
   3. Guessing game.
   4. Complex reaction time (not described in this paper).
   5. Post-Drug Interview.

from the shape and colour of the tablets administered. Very few of the subjects felt that they had received an active medication. There was no correlation between actual medication administered and the subject's evaluation of this medication. The use of oral drugs was adopted to facilitate recruiting subjects. The use of oral medication, however, precluded calculating dosage on the basis of body weight or other individual criteria.

(*c*) DETAILS OF TEST PROCEDURES USED

1. *The Stroop Test*

Out of all the various procedures used to assess the focus of attention, the Stroop (22) colour-word interference test is the simplest and the most readily administered (5). This test has been described by Thurstone (23) and more recently it has been used by Klein and co-workers (18), and Broverman and Lazerus (1) to measure factors which seem related to the focus of attention. In an unpublished pilot study alcohol was found to impair Stroop test performance, and alcohol would, on theoretical grounds, be expected to broaden attention.

In the Stroop test the subject is first given a card with 100 printed names of colours and asked to read off these words as rapidly as possible. He is then given a second card that has 100 spots tinted with the four primary colours, and asked to read these colours as rapidly as possible. The time required for this operation is recorded. Following this, he is given a third card and asked to name colours that have been tinted on top of the names of conflicting words. On this last card each colour name is tinted with a conflicting hue. For example, the word RED may be tinted yellow, green, or blue. The subject is instructed to read the actual colours and ignore the printed words. It generally takes considerably longer to read colours superimposed on conflicting names than it does to read colours alone (22, 23). If the subject is of above average intelligence and is more familiar with naming words than naming colours, then the conflicting names act as peripheral stimuli. The more narrowly the subject is able to focus his attention, the better he is able to filter out the interfering names and respond to the colours alone. The time taken to read colours on words, minus the time to read colours alone, gives a numerical score of colour/word interference.

2. *The Witkin Embedded Figures Test*

In Embedded Figures tests the subjects are first shown small figures and then shown larger designs in which the small figures are embedded and hidden. In each case the larger design serves as an interfering stimulus which must be ignored or filtered out before the subject can discover the hidden design. Atropine impaired performance on the Gottschaldt version of the Embedded Figures test.

Witkin and his group (24) have described a coloured modification of the Embedded Figures test. The Stroop test fails to discriminate between a narrowed focus of attention and a change from form to colour dominance. This does not seem an important objection, but the use of the coloured Embedded Figures test should help remove any doubts. For example, if a drug increased colour dominance, it should improve performance on the Stroop test but impair performance on a coloured Embedded Figures test, where colours appear as interfering stimuli.

The short form of Witkin's Embedded Figures test as described by Jackson (15) was used. This includes 12 of Witkin's original 24 figures. In administering this test, a subject is briefly shown a coloured complex design on a white card and told he will be asked to find a simple figure embedded within it, and that the simple figure will have the exact size, orientation, and shape of the one to be shown next. After these instructions the card is reversed, and on its back the subject sees a simple black line figure. He is asked to remember this and told he should try to find it hidden in the complex design on the other side without referring back. He is asked to inspect the simple figure until he is sure he remembers it, and then the card is turned over again. The subject is allowed two minutes to find the embedded figure without help. At the end of this time another card with the simple figure alone on it is held alongside the complex design, and an additional minute is allowed for finding the embedded figure with this help. Finding the embedded figure within the first two minutes rates a score of two. If part of the additional minute with help is required to find the embedded figure, a score of 1 is given. Total time required is recorded and each subject obtains two scores; a score for solutions and a score for time. For statistical handling, scores for solutions were ranked, and tied scores were broken on the basis of time.

3. *The Progressive Matrices*

The Progressive Matrices is a so-called culture free intelligence test designed by J. C. Raven commercially available in the United States from Western Psychological Services. In a previous study we found it to correlate well with the Embedded Figures test but to be relatively unaffected by atropine (4). It was felt worth while to include some control as to intelligence in this group of subjects, and pre-testing with the Progressive Matrices served this purpose. At the same time the pre-test Progressive Matrices gave a control for comparison with the post-drug Embedded Figures test, in the same way that two different types of Gottschaldt Embedded Figures tests were used in the atropine study. For statistical handling, ranks were assigned according to the number of correctly solved matrices, and tied ranks were broken on the basis of time.

4. *The Guessing Game as a Measure of the Focus of Attention*

In a previous study the Guessing Game was also used to measure the focus of attention (4). In this test the subject guesses which one of two lights will be turned on. In answer to each of the subject's predictions, one of the lights is turned on for a moment. After the subject observes the event he has attempted to predict, he guesses again; and again, after his guess, he observes. And so it proceeds, with guesses following observations, and observations following guesses. In a previous study the answer lights were shown with unequal frequency. Ordinarily, subjects are more likely to predict the more frequent light than they are to predict the less likely light. As compared with controls, subjects given methamphetamine (to narrow their attention) made fewer guesses for the more likely light and more guesses for the less likely light. This seemed to indicate that narrowing of attention produced patterns of guessing that were more independent of the lights the subjects were seeing, but this observation required additional study. In the study to be reported we had our subjects make predictions during a sequence of 120 light presentations. For the first 20 lights, the two lights were turned on with equal frequency. During the next 60 presentations, one light was turned on 80 per cent. of the time and the other light was turned on 20 per cent. of the time. During the last 40 light presentations this 80–20 ratio was reversed, and the previously infrequent light became the most frequent one.

RESULTS

Figure 1 shows mean change in Stroop scores, mean Embedded Figures solutions scores, and mean Embedded Figure times. The differences between all measures following medication are in the predicted direction.

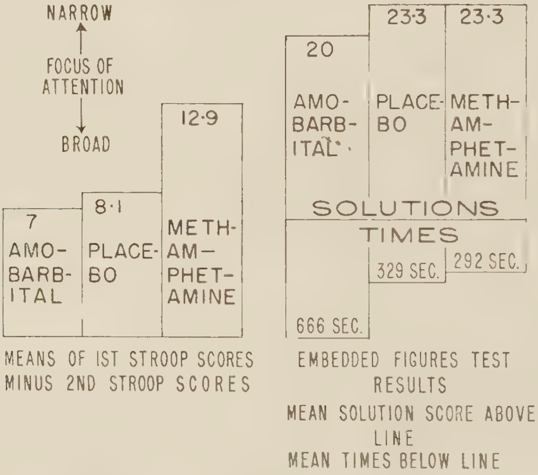

FIG. 1.—Summary of raw data.

To duplicate the earlier study on the effects of atropine (5), we computed a "change-in-ranks" score, as shown in Table I. Scores on first Stroop tests and scores on second Stroop tests were ranked separately. Then a difference in rank was taken so that a positive value indicated broadening of attention (worsening of Stroop score). In the previous study we had found a correlation between Progressive Matrices scores and Embedded Figures scores. We therefore ranked the subjects' performance on the Progressive Matrices tests as described and ranked scores on the Embedded Figures test as described. Again a difference was taken so that a positive score indicated an impairment in performance (theoretically, broadened attention). Then, following the procedure of the atropine study, the two rank scores were added, re-ranked, and tested for significance. Krushall-Wallace's (7) test yielded an "H" value of 11·705. "H" is distributed as chi square (with two degrees of freedom in this case), so the above value is significant at $P < \cdot 01$.

The results on the guessing game are summarized in Figure 2. We had predicted that methamphetamine treated subjects would make fewer guesses for the more frequent light while amobarbital treated subjects would make more guesses for the more frequent light. There is a tendency for the methamphetamine treated subjects to make fewer guesses for the more frequent light. As for the amobarbital treated subjects, the slight trend is not even in the predicted direction.

Table II shows the rank order correlations observed in this study. In a smaller pilot study correlations were found between the Stroop measure of narrowed attention and both introversion and neuroticism as measured on the Maudsley inventory. From the current data there would seem to be no significant relationship between neuroticism and measures of narrowed attention, and there are positive correlations between measures of narrowed attention and extraversion, which would be expected by chance less than one time in ten. This correlation is difficult to account for, since it runs counter to pilot study results. However, it would seem reasonable to discard any hope that there is a relationship between Eysenck's introversion factor and the concept of narrowed attention presented here.

TABLE II

Rank Order Correlations Between Measures: N=27

	Stroop No. 2	Embedded Figures	Extraversion	Neuroticism	Progressive Matrices
Stroop No. 1 ..	+·61	+·38	−·43	−·15	+·07
Stroop No. 2 ..		+·08	−·29	+·06	+·01
Embedded figures ..			−·41	−·10	+·20
Extraversion				−·07	+·13
Neuroticism					−·06

DISCUSSION

Using a combined score derived from performance on the Stroop test and on an Embedded Figures test, atropine (5) was found to produce changes consonant with broadening of attention. In the study reported above, methamphetamine was found to produce changes on an analogous combination score that indicated narrowing of attention. This is as predicted since methamphetamine had been found on earlier studies to produce other changes in performance indicative of narrowing of attention.

Amobarbital is known to have a depressing influence on the ascending reticular formation and we have postulated a relationship between activity in the ascending reticular system and the focus of attention. On the basis of this theory, amobarbital would be expected to produce a broadening of attention. In addition we had suspected a relationship between narrowing of attention and introversion and so further expected amobarbital to produce broadening of attention because Eysenck's group found it to have extraverting effects. As predicted, amobarbital is found to produce a broadening of attention on the combined Stroop-Embedded Figures score. Unfortunately, certain reservations are held about these results.

The combined score change with amobarbital is largely due to Embedded Figures performance. These Embedded Figures test results are skewed by a low optimum performance ceiling. Since the maximum solution score is 24, it can be seen in Figure 1 that the scores from this particular group of subjects clustered about the maximum performance end of the scale. Because of this, there was very little room for methamphetamine to produce improvement, and so the positive results with methamphetamine can be accepted with little reservation. On the other hand, the Embedded Figures tests were spuriously sensitive to impairment, and the apparent impairment induced by amobarbital is therefore open to question.

In addition to this, another problem was encountered. Previous experiments had shown significant correlation between Embedded Figures test performance and Stroop test performance. Table III shows that scores from the first Stroop test had a rank order correlation with the Embedded Figures test score of $+\cdot 38$. However, the second Stroop test score and the Embedded Figures test scores only correlated at $+\cdot 08$. This surprising result provoked us to do a scattergram,

TABLE III

	Difference Between Ranks of Scores				
Subject	1 (Stroop 1–Stroop 2)	2 (Progressive Matrices-Embedded Figures)		Combined Score (1 and 2)	Rank of Combined Score
1 ..	+ 1·0	−21		−20·0	27·0
2 ..	+ 4·5	− 5		− 0·5	15·0
3 ..	− 7·0	− 9		−16·0	25·0
4 ..	− 9·0	− 3		−12·0	21·0
5 ..	+ 4·0	+13	Control	+17·0	3·5
6 ..	+ 7·5	− 9		− 1·5	16·0
7 ..	+13·5	− 1		+12·5	5·0
8 ..	+ 2·5	+ 8		+10·5	6·0
9 ..	+ 3·5	−11		− 7·5	19·0
					137·5
10 ..	−13·5	− 1		−14·5	22·0
11 ..	− 5·0	+ 2		− 3·0	17·0
12 ..	−18·0	+12		− 6·0	18·0
13 ..	− 1·0	+ 5		+ 4·0	13·5
14 ..	+ 4·0	−19	Methamphetamine	−15·0	23·0
15 ..	+ 4·5	−16		−11·5	20·0
16 ..	+ 3·0	+ 1		+ 4·0	13·5
17 ..	+ 7·5	−23		−15·5	24·0
18 ..	− 2·5	−14		−16·5	26·0
					177·0
19 ..	− 5·0	+12		+ 7·0	11·0
20 ..	+ 4·5	+ 2		+ 6·5	12·0
21 ..	− 1·0	+18		+17·0	3·5
22 ..	+ 6·5	+ 1		+ 7·5	10·0
23 ..	+ 2·5	+ 7	Na. Amobarbital	+ 9·5	7·5
24 ..	+ 7·5	+ 2		+ 9·5	7·5
25 ..	+11·0	+ 5		+16·0	2·0
26 ..	−10·0	+18		+ 8·0	9·0
27 ..	+ 1·0	+25		+26·0	1·0
					63·5

which revealed that subjects with very low Stroop scores, that is, subjects with exceptionally narrowed attention, were doing poorly on the Embedded Figures test instead of doing well as was predicted. In other words, the distribution was "U" shaped. Re-inspection of the data from the atropine experiments showed that a tendency for the same "U" shaped relationship had been missed in going over that data originally. These findings are illustrated in Figure 2, where mean

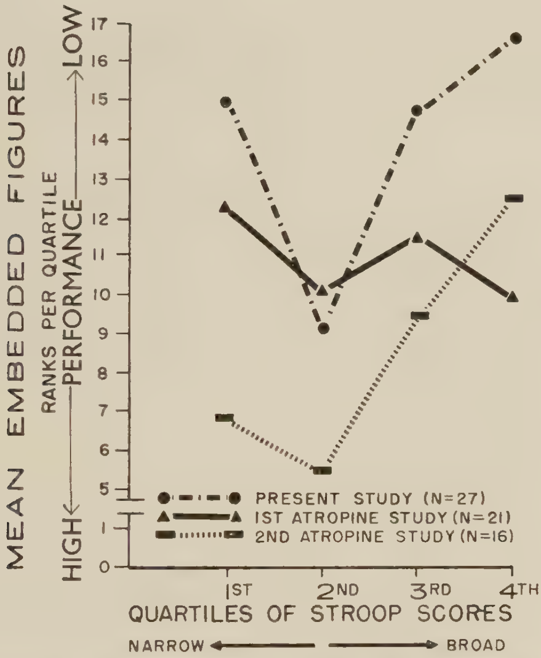

FIG. 2.—The relationship between Embedded Figures Test Performance and Stroop Test Performance.

Embedded Figures test ranks are shown for four Stroop quartiles. Because of the probability of the "U" shaped relationship between Embedded Figures scores and focus of attention, some subjects with actual narrowing of attention after amobarbital may perform poorly on the Embedded Figures test; again giving a spuriously low score for the amobarbital treated group.

As predicted, the methamphetamine subjects made fewer guesses for the more likely light and more guesses for the unlikely light. Although this observation could have occurred by chance, it is of some value since it repeats an earlier observation. The amobarbital treated subjects did not even show a change in the predicted direction. Performance on the guessing game is a rather complex affair (2) and it would appear that the effects of drugs on guessing game performance are weak. Theoretically, we would expect someone with a narrowed focus of attention to approach a task such as the guessing game with rather fixed preconceived strategies and his behaviour should be relatively independent of the particular pattern of lights that he might be observing. Put in other words, we would expect the person with narrowed attention to make his guesses less contingent on the lights that he was seeing in the guessing game. The response of guess frequency to a change in light frequency is delayed in methamphetamine treated subjects (Fig. 3) and may represent such a reduction in contingency of guesses on lights. Attempts to apply more elaborate measures of contingency (3) have so far been unsuccessful, although some promising leads are still being explored.

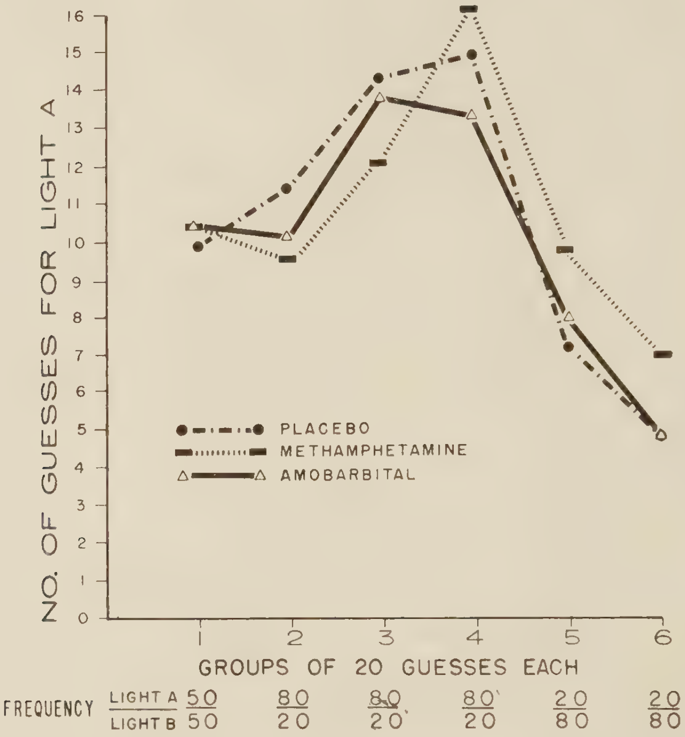

FIG. 3.—Performance on guessing game.

Failure to find the expected relationship between narrowed attention and introversion was surprising. Drugs found by Eysenck and his group to have introverting effects are also found to narrow the focus of attention. Also, at first glance one would feel that the concept of narrowed focus of attention was quite similar to the concept of introversion. In retrospect a number of possible reasons for our failure to find a relationship between narrowed attention and introversion can be found. In discussing sodium amytal and extraversion, Laverty (19) writes: "A particular pattern of uninhibited behaviour, often at variance with the normal and conventional practice of the subject, may be a constant or a recurrent response to alcohol or sedation. Attitudes may appear which are never or seldom expressed in the normal state. Strong affective expression may appear (be released), such as euphoria, expansiveness, resentment, anger or depression." Laverty fails to note that similar release phenomena may appear after the administration of stimulant drugs.

A second difficulty may be related to the frequency of "U" shaped relationships between various measures of stress or arousal. In the study just reported such relationships are found between Embedded Figures tests and the Stroop colour-word interference test. Although improved performance on the Stroop test usually goes with improved performance on Embedded Figures tests, people with very narrowly focused attention (who do extremely well on the Stroop test), may do rather poorly on the Embedded Figures test. "U" shaped relationships between performance and arousal have been recently discussed by Stennet (21), and by Cohen *et al.* (7) and may be considered a logical extension of the Yerkes-Dodson "law" (25). Consequently, it would seem that clusters of traits derived by the factor analysis of linear correlations would not include certain psychological phenomena related to the neurophysiological phenomena of arousal.

Items used in the Maudsley Personality Inventory to measure introversion/extraversion illustrate a third point. Although Eysenck and his co-workers rely heavily on the Jungian concepts of extraversion and introversion, especially as modified by Gilford, they fail to take much account of sensitivity to environmental stimuli.

The introvert may be considered reserved or withdrawn, but classically he is also considered somewhat free from the immediate demands of the environment. A group of personality items were found by Schlesinger (20) to correlate with susceptibility to interference, and this "susceptibility to interference" would seem very close to "focus of attention". A comparison between certain Maudsley items supposed to indicate extraversion and certain items found by Schlesinger to correlate with susceptibility to interference is given in Table IV to make the differentiation between the two concepts clearer.

TABLE IV

Maudsley Personality Inventory Items Indicating Extraversion	Items Found by Schlesinger to Correlate with Non-focusing (i.e. High Susceptibility to Interference or Broad Attention)
2. Do you prefer action to planning for action? ..Yes.	1. Sometimes I talk myself into feeling the way I should. ..False.
6. Are you inclined to be quick and sure in your actions? ..Yes.	2. My spirits depend very much on my surroundings. A gloomy room makes me feel that way too. ..True.
46. Would you be very unhappy if you were prevented from making numerous social contacts? ..Yes.	3. I am easily moved to tears by a sad part in a movie. ..True.
61. Would you rate yourself as a lively individual? ..Yes.	4. My feelings are easily hurt by ridicule or by the slighting remarks of others. ..True.

It can be seen that the Maudsley Personality Inventory items for extraversion deal with activity in the environment as opposed to a more introspective way of life. On the other hand, the susceptibility to interference items deal with sensitivity to intrusive affects and perceptions, both from within and without. This contrast seems worth some attention, although the relative merits of each concept can only be evaluated on the basis of further empirical studies.

There was only one point in which the above data seemed related to the Eysenckian dimensions of personality. Laverty (19) reported that neurotic introverts showed the most marked changes towards extraversion following sodium amytal. In the experiments reported here, no particular relationships between extraversion and response to amobarbital were found. However, subjects who scored highest in neuroticism on the Maudsley Inventory showed the greatest change towards broadness of attention following amobarbital.

In spite of these negative findings, it seems logically difficult to assume that the focus of attention and the continuum extraversion/introversion have no relationship at all. Since the focus of attention is a useful concept in predicting the effects of certain centrally active drugs upon behaviour, the exact relationship between the focus of attention and the continuum of extraversion and introversion demands additional study.

Summary

1. Using a combination of the Stroop colour-word interference test and Witkin's coloured Embedded Figures test to assess the focus of attention, (*a*) methamphetamine was found to narrow attention, and (*b*) amobarbital was found to broaden attention.

2. A theoretical basis for expecting a correlation between measures of narrowed attention and the Maudsley Inventory measure of introversion is presented. The data, however, fail to bear out such a supposition since observed correlations between measures of narrowed attention and introversion were significantly negative.

3. The significance of the above findings, as well as certain reservations, are discussed.

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HOMOSEXUALITY AMONG OXFORD UNDERGRADUATES[†]

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In Oxford, all psychiatric cases among undergraduates are referred by their general practitioners and therefore tend to be selected by them. There is no group they are more ready to pass on to the psychiatrist than the student complaining, in whatever guise, of homosexuality. It is not therefore surprising that homosexuals form a high proportion of the 100 Oxford students referred on psychiatric grounds and studied in the research underlying this paper (the mode of selection is described in Davidson *et al.*, 1955 and Spencer, 1957, p. 93). Opportunity was afforded also for the examination of 100 freshman controls from three Oxford Colleges (for mode of selection, see *loc. cit.*). These students agreed to answer questions about their sexual development, including possible homosexual practices or interests.

The data come from these two groups.

TABLE I

Subjects Displaying Homosexual Practices, Phantasies or Desires

	Patients No. = 100	Controls No. = 100	Correlations (+ve Between Patients and the Criterion)
Total homosexual	51	35	r = ·27*
Grown out of	10	27	r = ·40**
Persistent	41	8	r = ·64**
Prognosis guarded ..	24—58·5%	Prognosis not Estimated	
Prognosis poor	10		
Prognosis dubious ..	14		
Prognosis good	17—41·5%		
Practices only	27	13	r = ·32*

INCIDENCE

Table I shows the bare figures for incidence of homosexual interest and practice in the 2 groups. The total of homosexual patients is significantly greater than that of the controls, and so is the total of homosexuality practising patients compared with that of the controls. Not one of the 13 practising controls admitted a continuation of practice, but 3 of them agreed that the urge to it persisted. However, one of the potential controls was excluded when found to be under care for inverted practices (see discussion, below). One control with persistent trends later broke down and came into hospital.

[†] Based on part of a thesis submitted in 1957, for the M.D. of Oxford University and on a memorandum submitted by the British Psychological Society as part of its evidence before the Interdepartmental Committee on Homosexual Offences and Prostitution. The memorandum was first put before a working party of the Society, of which I was a member.

Throughout the paper, * indicates statistical significance by χ^2 (Yate's modification used where necessary) at the 5 per cent., and ** at the 1 per cent. level. All correlations, unless otherwise stated, are tetrachoric.

LACK OF HETEROSEXUAL INTEREST

Lack of heterosexual interest was by no means synonymous with the presence of homosexual trends in the controls. Of the 18 patients lacking heterosexual interests, all but one possessed homosexual ones (r=·77**), but only 1 of the 7 controls displaying no heterosexual interests admitted to homosexual ones (r=—·37).

PARTNERS

A non-practising homosexual will have a partner in phantasy. Table II, then, displays age, relative to the patient, of the partner, phantasied or real, of all the homosexuals, both patients and controls; and that of real-life partners of the practising homosexuals. In both patient and control groups, in phantasy and in fact, the partnerships were mainly among coevals. The significant increase in relations with older men among the patients is interesting, as this form of relation is likely to be more passive and "feminine".

TABLE II

Ages, Relative to Subjects of Real or Phantasied Homosexual Partners

	Older	Same Age	Younger or Boys
		(*a*) All Homosexuals	
Patients No.=51 ..	12—23·5%	44—86%	16—31·5%
Controls No.=35 ..	2— 5·7%	28—80%	13—37%
Correlation	r=·48*	—	—
		(*b*) Practising Homosexuals	
Patients No.=27 ..	9—33·3%	24—89%	8—29·5% (including 3 boys)
Controls No.=13 ..	1— 7·5%	11—85%	3—23%
Correlation	r=·53 (not significant)	—	—

N.B.: One subject may have 2 types of partner.

FORM OF PRACTICE

Table III shows the main forms of relations practised by the patients and controls concerned. Mutual masturbation is far the commonest form used by

TABLE III

Form of Homosexual Practice

	Mutual Masturbation			Other	
	Active	Mutual	Passive	Sodomy	Fellatio
Patients No.=27	13—48%	12—44·5%	8—29·5%	2—7·5%	1—3·5%
Controls No.=13	—	10—77%	4—36%	—	—
Correlation ..		r=—·50			

both groups—exclusively that of the controls, not one of whom took the active role alone and few the passive only.

Physique

Both total groups correlated positively with mesopenia or muscle lack, as Figures 1 and 2 show. From Figure 3, the trend can be seen still more markedly in the 24 patients of uncertain prognosis.

Taking mesopenia to refer to mesomorphy of 3 or less on Sheldon's 7-point scale, the numbers and correlations against the rest of the patients or controls are:

Of the 51 patients, 25 had mesopenia, r= ·32*.
Of the 35 controls, 21 had mesopenia, r= ·42**.
Putting the 2 together (there were 39 mesopenic patients altogether, 42 controls), r= ·35**.
Of the 24 patients of uncertain prognosis, 16 had mesopenia, r= ·51**.

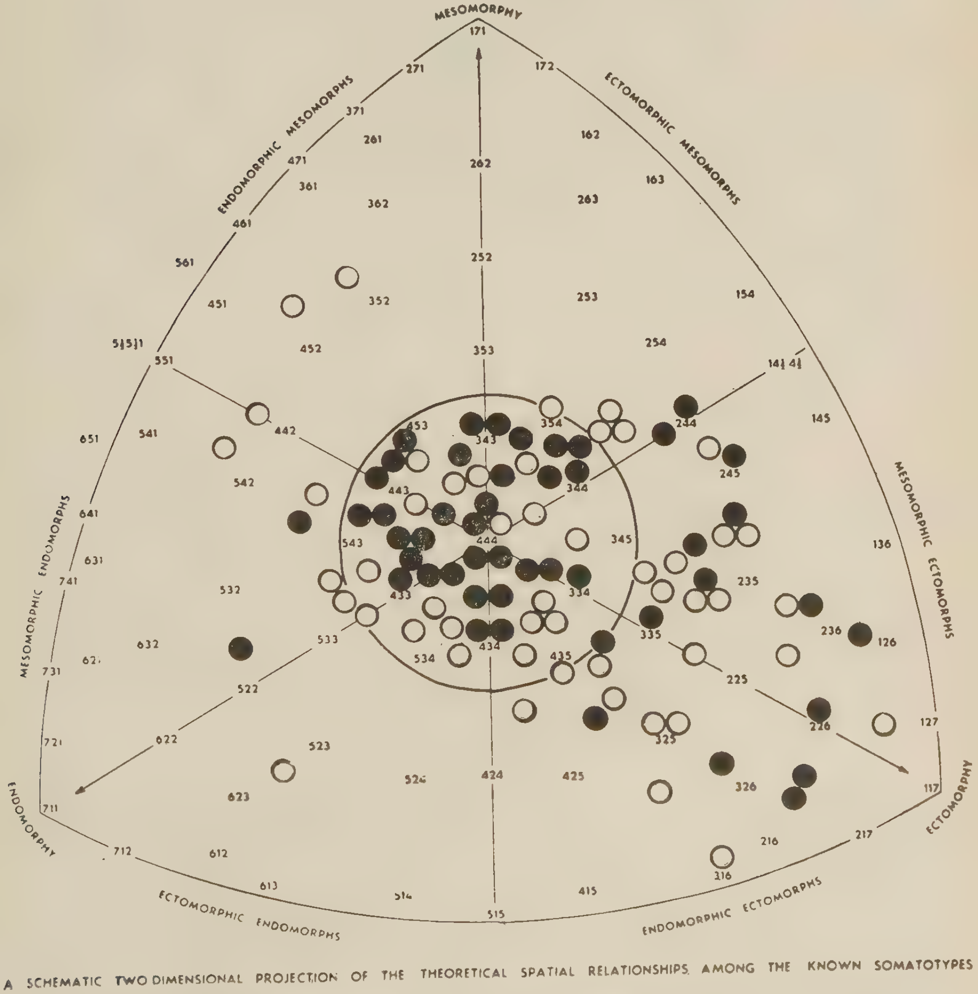


FIG. 1.—Somatotype distribution of 100 Oxford undergraduate psychiatric patients.
○=51 homosexuals. ●=rest of group.

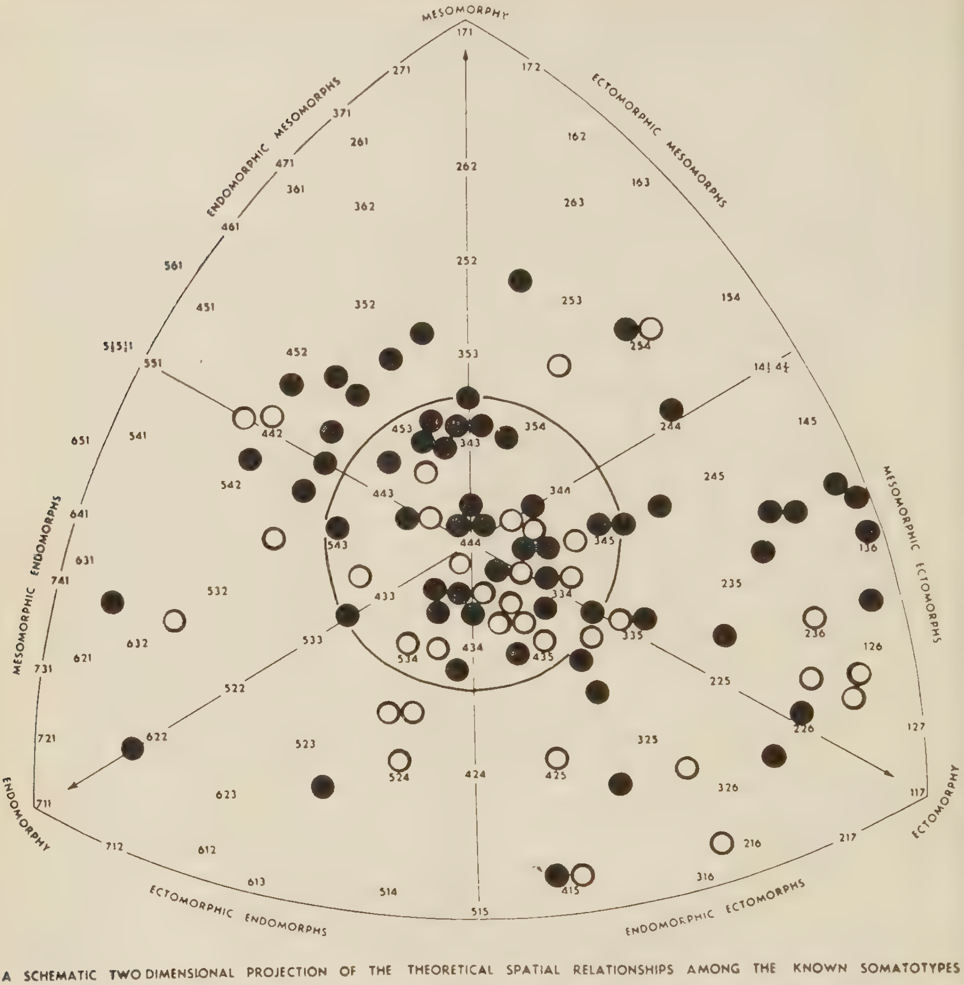


FIG. 2.—Somatotype distribution of 100 Oxford undergraduate volunteer controls.
○=35 homosexuals. ●=rest of group.

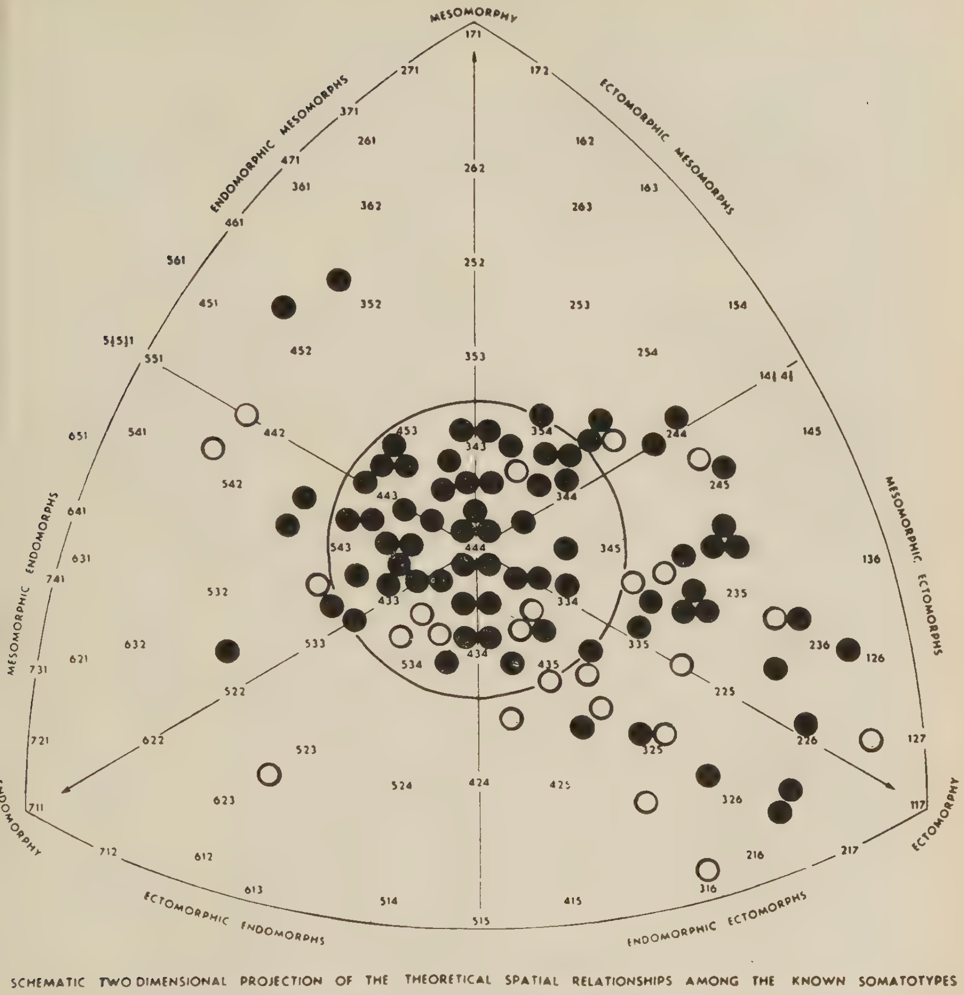
On the other hand, the 10 patients who had completely lost their homosexual interests were inclined to mesomorphy at the level of 3^2 or more (r= ·11).

As Figures 1 and 2 show, both the homosexual patients and controls tended to ectomorphy: but not significantly (rs respectively= ·28 and ·24).

PERSONALITY

A general assessment of stability of personality was made in both groups on a 4-point scale (++, +, ± and —). Stability was defined as "well adjusted within self and to University life as judged by history and interview" (Spencer, 1957, p. 131).

In Table IV, the stability of both homosexual groups is shown in comparison with their parent group and with each other. It will be observed that whereas, among the patients, persistence and poverty of prognosis go with increasing instability, among the controls the trend is slightly in the adverse direction.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ECTOMORPHIC ENDOMORPHS
ENDOMORPHIC ECTOMORPHS
SCHEMATIC TWO DIMENSIONAL PROJECTION OF THE THEORETICAL SPATIAL RELATIONSHIPS AMONG THE KNOWN SOMATOTYPES

FIG. 3.—Somatotype distribution of 100 Oxford undergraduate psychiatric patients.
○=24 homosexuals of uncertain prognosis. ●=rest of group.

TABLE IV

Instability and Homosexuality

	++	+	±	−	Correlations ++ or +
All patients (No.=100) ..	20	37	33	10	
All homosexual patients (No.=51)	13 (25·5%)	21 (41%)	14 (27·5%)	3 (6%)	
Persistently homosexual patients (No.=41)	11 (27%)	17 (41·5%)	12 (29%)	1 (2·5%)	v. Rest of patients, r=·30
Homosexual patients with dubious prognosis (No.=24)	10 (41·5%)	9 (37·5%)	5 (21%)	—	v. Rest of patients, r=·49*
All controls (No.=100) ..	4	13	38	45	
All homosexual controls (No.=35)	1 (3%)	3 (8·5%)	17 (48·5%)	14 (40%)	v. All homosexual patients, r=−·80**
Homosexual controls with persistent trends (No.=8) ..	—	1 (12·5%)	5 (62·5%)	2 (25%)	v. Persistently homosexual patients, r=−·73*

MINNESOTA MULTIPHASIC PERSONALITY INVENTORY

Any Sub-category

It was found that only 23 out of 93 tested patients scored less than 70 on any sub-category whereas only 24 out of 89 tested controls scored more (r= ·69**). Taking 70 or more, then, as criterion of "instability" on this test, both patients and control homosexuals emerged very slightly but insignificantly "unstable" (respective rs= ·03 and ·15).

Mf-scale

This scale is supposed to measure feminine interests in males. Table V shows that it only does so crudely in these groups. Among the controls, the scores of the homosexuals do not differ from those of the remainder. There is a slowly rising score among the patients as one traverses the "degrees" of homosexuality; but a significant contrast with the rest is achieved only by a small group of homosexual patients of particularly poor outlook and on the score of 80+. That this sub-category measures some form of instability is shown by the marked difference in the patients' from the controls' scores, the correlation at the 80+ level approaching that for any sub-category at the 70+ level.

TABLE V

MMPI—mf Scale

	60+	70+	80+	Correlations
All patients (No.=93)	75 (81%)	46 (49·5%)	20 (21·5%)	
All homosexual patients (No.=48) ..	41 (85·5%)	25 (52%)	12 (25%)	
Persistently homosexual patients (No.=38)	33 (87%)	21 (55%)	11 (29%)	
Homosexual patients of guarded prognosis (No.=22)	22 (100%)	14 (63·5%)	8 (36·5%)	
Homosexual patients of poor prognosis (No.=9)	9 (100%)	7 (79%)	6 (66·5%)	70+, v. rest of patients, r= ·43 80+, v. rest of patients, r= ·65**
All controls (No.=89)	54 (60·5%)	13 (14·5%)	2 (2%)	60+, v. patients, r=—·30** 70+, v. patients, r=—·58** 80+, v. patients, r=—·65**
All homosexual controls (No.=32) ..	16 (50%)	6 (19%)	2 (6%)	
Persistently homosexual controls (No.=7)	5 (71·5%)	2 (28·5%)	—	

BACKGROUND FACTORS

The only background factor in Table VI, with which homosexuality appears associated, is previous psychological illness: and then only among the patients since but 2 homosexual controls ever had such a breakdown. As the previous breakdown, in 9 of the 12 patients in question, involved the complaint of homosexuality, the general implication, that the patients' homosexuality was of a more disturbing type than the controls', is confirmed.

NUMBER OF SIBLINGS

Lang (1940) claims that the siblings of homosexuals, instead of dividing into the habitual 105 males to 100 females, gravitate in fact into a ratio of 120 males to 100 females. He hypothesizes from these findings that the homosexual is genetically female.

TABLE VI

Background Illness and Relations

	Family Mental Illness	Previous Mental Illness in Subject	Disharmony Between Parents or Broken Home
All patients (No.=100)	40	17	47
All homosexual patients (No.=51)	22—43% r=·10	12—23·5% r=·33 (nearly significant)	25—49% r=·07
Persistently homosexual patients (No.=41)	19—46·5% r=·18	11—27% r=·40*	19—46·5% r=—·03
All controls (No.=100)	19	2	28
All homosexual controls (No.=35)	7—20% r=·03	1—3% —	12—34% r=·18
Persistently homosexual controls (No.=8)	3—37·5% r=·31	—	2—25% r=—·05

Taking all the homosexuals, patients and controls together, it was found here that the entire group had a total of 81 brothers to 66 sisters, a ratio of 125 to 100, agreeing exactly with Lang's (but not significant, χ^2 being 1·0).

When this total figure is broken down, however, it is found that, whereas the homosexual patients had 42 brothers to 44 sisters (95:100) the controls had 39 brothers to 22 sisters (170:100, $\chi^2=3{\cdot}8$, on verge of significance). Since evidence already cited makes it plain that the degree of homosexuality was more deep-set in the patients, it is difficult to see how this distribution can be accorded with Lang's hypothesis: the patients, more persistently and disturbedly homosexual, should have been genetically more female and therefore have had more brothers. Furthermore, the 27 controls and 10 patients "swung" over to complete heterosexuality, taken together, have 43 brothers to 26 sisters (170:100, $\chi^2=3{\cdot}3$). The significance of this is discussed later.

SCHOOLING AND SOCIAL CLASS

Here occurs another anomaly lacking ready explanation. From Table VII it can be seen that (1) the proportions of all patients, all controls and all homosexual patients at day and boarding schools, were nearly identical; (2) the homosexual controls (most of whom claimed to have "swung" into total heterosexuality) and those of them and of the patients who did so claim, correlated positively and significantly with *boarding* school education; and (3) the majority of both persistently homosexual groups were *day* schoolboys.

As the patients and controls are so mutually consistent in this respect, they have been put together at the end of the table and the figures show the marked preponderance of *day*-school education among the persistent homosexuals, of *boarding* education among the "swung over". This difference is discussed later. It is not simply due to the persistent homosexuals being of lower social class than the "swung" over and therefore educated at state schools. There was no significant correlation between any form of homosexuality, among patients or controls, and the paternal occupation being below the Registrar General's Social Classes I and II: 4 of the 8 persistently homosexual controls had fathers working in Class III or lower occupations, but the correlation of ·40 is not significant. The *tendency*, however, was for the persistently homosexual of both groups to come from the lower social classes, the "swung"-over from the upper.

TABLE VII

Type of Schooling

	Day	Boarding	Correlations (+ve When Higher Proportion at Day School)
All patients (No.=100) ..	59	41	
All homosexual patients (No.=51)	30 (59%)	21 (41%)	v. Rest of patients, r=0
Patients "swung" to heterosexuality (No.=10)	1 (10%)	9 (90%)	v. Rest of patients, r=—·70** v. Rest of homosexual patients, r=—·79**
Persistently homosexual patients (No.=41) ..	29 (71%)	12 (29%)	v. Rest of patients, r=·32* v. Rest of homosexual patients, r=·79**
All controls (No.=100) ..	60	40	
All homosexual controls (No.=35)	16 (45·5%)	19 (54·5%)	v. Rest of controls, r=—·35*
Controls "swung" to heterosexuality (No.=27)	10 (37%)	17 (63%)	v. Rest of controls, r=—·45** v. Rest of homosexual controls, r=—·53
Persistently homosexual controls (No.=8) ..	6 (75%)	2 (25%)	v. Rest of controls, r=·22 v. Rest of homosexual controls, r=·53
All subjects (No.=200) ..	119 (60%)	80 (40%)	
All homosexual subjects (No.=86)	46 (53·5%)	40 (46·5%)	v. Rest of subjects, r=—·19
Subjects "swung" to heterosexuality (No.=37)	11 (30%)	26 (70%)	v. Rest of subjects, r=—·51** v. Rest of homosexual subjects, r=—·60**
Persistently homosexual subjects (No.=49) ..	35 (71·5%)	14 (28·5%)	v. Rest of subjects, r=·40** v. Rest of homosexual subjects, r=·60**

RELATIONS OF SUBJECTS WITH PARENTS

Relations of all subjects with each parent were classified as harmonious, mildly disturbed and more severely disturbed (see Davidson *et al.*, 1955, Table VI). As only 2 persistently homosexual controls were disharmonious with their fathers and one with his mother, the homosexual controls have not been subdivided in Table VIII. The important feature is the significant correlation of homosexual patients and persistently homosexual patients with a disharmonious relation with the mother, provided both degrees of disharmony were included. The homosexual controls showed a tendency also to this disharmony. The proportions disharmonious with the father varied remarkably little, throughout both the patients' and controls' homosexual range, from those of the parent group.

The anomalous finding is the high proportion of "swung" homosexual patients in severe discord with their mothers: nearly half those thus disturbed are from this group. I can offer no explanation of it.

TABLE VIII

Relations With Parents

	With Father			With Mother		
	Mildly Disturbed	More Severely Disturbed	Total Disturbed	Mildly Disturbed	More Severely Disturbed	Total Disturbed
All patients (No. = 100)	26	16	42	30	11	41
All homosexual patients (No. = 51)	14 (27·5%)	8 (15·5%)	22 (43%)	18 (35%)	9 (17·5%) r = ·47*	27 (52·5%) r = ·39**
Patients "swung" to heterosexuality (No. = 10)	3 (30%)	1 (10%)	4 (40%)	—	5 (50%) r = ·70**	5 (50%)
Persistently homosexual patients (No. = 46)	11 (27%)	7 (17%)	18 (44%)	18 (44%)	4 (10%)	22 (54%) r = ·33*
All controls (No. = 100)	9	2	11	4	3	7
Homosexual controls (No. = 35)	3 (8·5%)	1 (3%)	4 (11·5%)	3 (8·5%)	1 (3%)	4 (11·5%) r = ·30

Only significant or nearly significant correlations compared with the rest of the parent group are given.

FURTHER GROUPINGS OF PATIENTS

(a) *Dynamic*

The patients were known much better than the controls. It was therefore feasible to differentiate the patients into the following divisions:

	Number
Group A—homosexuality of little importance in total syndrome	33
Group B—homosexuality of importance in total syndrome	18
Total	51

Sub-groups of Group A:

			Number
Sub-group I	Homosexuality grown out of		10
Sub-group II	Homosexuality mild and nearly grown out of in personalities mildly schizothymic		4
Sub-group III	Homosexuality playing subsidiary role in severe personality disorder		19
	(*a*) Personality severely schizothymic	9	
	(*b*) Personality extraverted with hysterical or psychopathic trends	10	
Total			33

Sub-groups of Group B—Constitutional and psychodynamic interplay:

		Number
Sub-group IV	"Pure culture" constitutional homosexuality	2
Sub-group V	Homosexuality associated with obesity suggesting endocrine dyscrasia	3
Sub-group VI	Constitutional factor +, psychodynamic factor ±	5
Sub-group VII	Constitutional factor ±, psychodynamic factor +	5
Sub-group VIII	"Pure culture" psychodynamic homosexuality	3
Total		18

From this sub-grouping, which is a clinical and therefore subjective assessment, it will be seen that a psychodynamic factor was discovered in 13 cases. Their analysis is as follows:

	Number
Mother died before adolescence	3
Mother rejecting, wholly or ambivalently	4
Total mother loss	7
Mother domineering	5
Mother sided with by patient in severe parental discord	1
Total over-identification with mother	6

No similar clear identification or rejection could be discovered in the relations with the father. This accords with the statistical correlation, shown in the section above, with disturbed relations with the mother but not with the father.

(b) *Kinsey Grouping*

In their heterosexual/homosexual rating scale (Kinsey *et al.*, 1948, pp. 639–41), Kinsey and his colleagues allow for homosexual "reactions" in place, if necessary, of homosexual activities (*loc. cit.*, p. 647). Where, they explain, the "overt sexual experience" and "psychosexual reactions . . . are not in accord . . . the rating of an individual must be based upon an evaluation of the relative importance of the overt and the psychic in his history" (*loc. cit.*). This has here been done:

		Kinsey Equivalent		*Number*
Group 1	Grown out of	1–2		10
Group 2	Nearly grown out of	2–3		9
Group 3	Hetero/homosexual interest about equal			11
	(*i*) Good chance of "swing" to full heterosexuality	3–4	8	
	(*ii*) Chance of "swing" over poor	4–5	3	
Group 4	Heterosexual interest small	4–5		4
Group 5	Heterosexual interest nil			17
	(*i*) Chance of "swing-over" to heterosexuality	5	7	
	(*ii*) Little chance of "swing-over"	6	10	
Total				51

This would make the Kinsey groupings summarize as:

0	1–2	2–3	3–4	4–5	5	6	*Total*
49	10	9	8	7	7	10	100

SUICIDAL ATTEMPTS

No patient was lost by suicide but 2 came under care first after severe suicidal attempts, 1 made a vague attempt in an acute psychotic episode, 5 made suicidal gestures, 1 came under care because of fear of doing himself harm, 1 as a result of threatening it histrionically. The total in this group is therefore 10.

Nine of these 10 were persistently homosexual in trend ($r=\cdot69^{**}$).

One of those making a severe attempt was rejected by his partner of desire. The patient fearful of it felt guilty following a homosexual episode on holiday with a friend and resentful of the subsequent cooling of the friendship. The friend of the histrionic threatener had refused to live with him at the end of their University careers. In the others, the relation of the suicidal and homosexual factor was less direct.

THERAPY AND PROGNOSIS

The prognostic assessment has already been shown in Table I. It will be seen that, of those with persistent trends, the prognosis for "swing over" was considered good in almost half.

In 5 cases only, it was felt that conspicuous help was given the patient by psychotherapy directed specifically to the homosexual factor. Of these, one patient, after making a valiant heterosexual attempt despite conspicuous effeminacy of manner, later entered a monastic order and has tended to relapse.

DISCUSSION

Controls and Incidence

Even though the figures do not coincide exactly with those submitted here, the reference in paragraph 40 of the "Wolfenden Report" to the 100 male undergraduates who provided "the only figures relating to the systematic examination of anything like a 'normal' (homosexual) sample" is to the control sample of this paper (Wolfenden, J., *et al.*, 1957). The recent factual study of Hemphill *et al.* (1958) was carried out on 64 homosexual prisoners in Bristol. It may therefore be of more than ordinary interest that 35 per cent. of a group of 100 "normal" Oxford freshmen admitted to some sort of homosexual interest, 13 per cent. to some type of practice and 8 per cent. to persistence of the trend. None admitted to persistent practice: even allowing, however, for deception, it may be that the group was examined too early in its undergraduate career to have been initiated into homosexual coteries the existence of which we have been led clearly to recognize. Moreover, the 100 of the research were selected by methods already described (Davidson *et al.*, 1955) from an original 130 subjects, one of whom was rejected on the ground that he was already under other psychiatric care for homosexual practice. If, then, 3 of the 8 controls, admitting persistence of homosexual interest, were to take up practice, this would make a total of 4 practising homosexuals out of 130—equivalent, in a University of 7,000 men, to over 200 such practising inverts. Whether the real incidence is as formidable, is conjectural: at least these figures suggest the possibilities of the problem in a residential and predominantly male University like Oxford: and 4 per cent. is the orthodox figure for the incidence of fixed homosexuality.

The Two Patterns of Inversion

The figures, here subtended, suggest strongly two distinct patterns of inversion, the first common and seen mainly among the controls, the second affecting 8 per cent. of controls, occurring in 41 per cent. of patients and being the major factor in the breakdown of 18 per cent.

Pattern 1 appears predominantly among the reasonably stable (controls, mostly) during adolescence (when, as Kinsey *et al.* showed (1948, p. 219), sexual potency is high) and is fading or faded by the time University life is reached. It does not cause breakdown and seems facilitated by the presence of brothers at home (though I have no figures of the incidence of inter-sibling mutual masturbation) and of other boys at boarding schools. It leads to practice mainly with coevals or, to a less extent, with younger boys. Interestingly, in this group, it occurred among "normals" of muscle-lacking physique and with only a tendency to maternal conflict, but among muscular patients in severe conflict with their mothers. It is as if constitutionally weak "normals" go through this phase which likewise is found among certain patients breaking down on other grounds: conflict with the mother may be aetiologically relevant to both the breakdown and the homosexuality.

One wonders from this evidence whether Lang (1940) was not naive in adducing genetic intersexuality from his finding of an increased proportion of brothers among his homosexuals. Was it not more probable that the presence of these brothers afforded the potentially homosexual an easier inroad into homosexual practice?

One wonders, too, if this group does not make-up the "facultative homosexuals" of wartime, ships and other all male situations.

Pattern 2, more often causing breakdown, exists among diverse physiques but appears of graver import for eradication among those of weak musculature. Being deeper-seated, it does not need the facility of brothers or fellow boarding schoolboys for its emergence, and is more frequently practised with older men. Presumably, indeed, some member of this group will become these "older men" in due course. As expected, this pattern is more frequently associated with disorder of personality, and once again—indeed more prominently—disturbed maternal relations supply the ascertainable dynamic factor. The 18 predominantly homosexual patients suggest that the constitutional/dynamic interplay occurs upon a relatively normal distribution scale, the mixture of both ingredients most often leading to homosexuality: the figures are too small to prove this. This group contributes strongly to the ranks of undergraduate suicides.

Physique

With acknowledgment to West's (1955) searches into the literature, I have elsewhere (Spencer, 1957, pp. 60–63) demonstrated the paucity of evidence for a constitutional factor, let alone one of physique, in homosexuality: but the figures here suggest that muscle-lack has a part to play in both patterns—in the transient and "normal", and in the dangerously persistent, groups. It looks as though a weak physique helps the mildly liable to succumb and the severely liable to become radically immersed; but is not the factor producing the liability.

Dynamic

The relation with the mother seems the potent dynamic factor, but its position here is anomalous. It exists a little among the controls and more strongly among the patients. The more severe the disturbance with the mother, the more frequent is the association with homosexuality; but the highest incidence of this severe disturbance occurred among the patients "swung" over. With this proviso, which greater numbers might not confirm, the view of the authorities cited by West (1955, p. 92), that psychogenic factors exceed constitutional in importance, is supported.

CONCLUSION

In the psychiatry of undergraduates, the importance of "Pattern 2" homosexuality, and its distinction from the innocuous "Pattern 1", cannot be overstressed. It was the major factor in the breakdown of 18 patients, brought one to a serious suicidal attempt and 2 near to it; while at least 2 successful suicides, never under care, were committed after homosexuality had come to light.

The mysteries of homosexuality are still so pervasive that all new facts must throw light. Here, the relation of mesopenia with maternal dynamics on the one hand, and on the other, the facultative factors in "Pattern 1" of brothers and boarding school, may help illuminate a small corner of this enshrouded field.

SUMMARY

1. Figures are given for the incidence and persistence of homosexual trends and practices among 51 of 100 Oxford undergraduate psychiatric patients and 35 of 100 freshmen controls. In all these incidences, the patients significantly exceeded the controls. In 18 patients homosexuality was the main cause of breakdown.

2. Lack of heterosexual interest corresponded with homosexual interest in the patients but not the controls.

3. Ten patients, 27 controls showed "pattern 1" of homosexuality in which an adolescent phase of interest in or practice with co-evals occurred in subjects having more brothers than average or going to boarding schools. Their stability was relatively high. The patients were relatively mesomorphic but in severe conflict with their mothers: the controls were mesopenic and in less maternal conflict. They may be "facultative" homosexuals.

4. Forty-one patients, 8 controls displayed "pattern 2" wherein the greater the muscle lack, the worse the prognosis. Constitutional and dynamic factors appeared along a normal curve of distribution among the patients. The group consisted mainly of day pupils. The controls tended to be in the lower social grades. The patients tended to relations with older men more than the subjects of "pattern 1".

5. Among the patients, at least 24 or 47 per cent. of all homosexuals were estimated to come into Kinsey gradings of 4 or more. In 14 the prognosis for "swing" over was considered dubious, in a further 10 definitely poor. Homosexuality was associated among the patients with suicidal attempts, gestures or urges. Psychotherapy was considered helpful in 5 patients.

6. The importance in these subjects of mesopenia and conflict with mother is stressed.

7. Lack of heterosexual interest was synonymous with homosexuality among the patients but not among the controls.

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THE PSEUDO-MENTAL-DEFICIENCY SYNDROME

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CLINICAL conditions simulating various degrees of intellectual handicap have been described quite frequently in the past. Individuals with such a deficiency have been observed in increasing numbers by psychiatrists, pediatricians, psychologists and social workers in schools for the mentally defective as well as in psychiatric hospitals and mental hygiene clinics.

The syndrome of pseudo-mental deficiency has been investigated by improved methods of psychological examination and by the increased skill and experience of psychiatrists and allied professional personnel. Pseudo-mental deficiency as used in psychological literature during the past fifteen to twenty years is almost as diffuse and broad a term as the designation of mental defect itself. Wide and extensive knowledge is therefore needed in the field of psychological medicine and pediatrics if the two clinical groups of mental deficiency, pathological and the functional, are to be held apart correctly and differentiated clinically. Any considerable delay in the prompt recognition and diagnosis of the underlying causes of these two states of intellectual insufficiency may adversely affect any subsequent counselling and rehabilitative efforts of either of these two clinical groups. The pseudo-defectives if left untrained and untreated too long, would no longer be capable of catching up with their fellows. The true mental defectives, on the other hand, would gradually become more difficult to train, and would eventually develop into problem persons or drift into anti-social patterns of behaviour.

The factors most commonly responsible for the incidence of pseudo-mental deficiency can be attributed to causes inducing a delay in physical, mental and affective maturation, defects or embryological anomalies in specific sense organs, emotional and environmental deprivations, psychoneurotic and psychotic states based on functional or organic pathology. Conditions predisposing to pseudo-retardation and chronological lag in mental and educational growth are of considerable aetiological importance and will be discussed accordingly.

There appear to be a considerable number of variations in time at which educationally normal children reach a maximal phase at which they are capable of functioning adequately to a standard curriculum of education. Other agencies such as interpersonal relationship, level of socio-economic standing, attention span and physical well-being, may to a certain extent determine the scholastic readiness of the pupil. Should any of these handicaps constitute a serious drawback in the make-up of such a child, he would certainly find it increasingly difficult to profit from a stereotyped, rigidly applied school programme. As a consequence, one would not be justified in attributing a child's slow educational progress or faulty social adjustment primarily to a basic insufficiency of innate mental endowment.

The spastic child as an example belongs in this category. On an average he develops remarkably slowly, but often continues to grow intellectually beyond the expected level of mental maturation of a defective child who has no orthopaedic handicap. As a matter of fact, not infrequently his mental growth keeps developing throughout adolescence, often surpassing the average level of maturational increase of normal children.

There are also a number of other physical and mental disorders which may lead to a delay of intellectual development. The clinical conditions referred to comprise certain forms of tuberculosis, the early infantile forms of aberration, some types of infantilism, improper nutritional and vitamin deficiencies and other morbid states such as rickets, chronic infantile intestinal diseases and diverse serious chronic bodily ailments.

A lack of sensory acuity in one or more of the specific sense receptive organs often conveys the false impression of such persons being dull or mentally retarded. They may not be able to see or hear as well as their classmates which in turn may tempt the teacher to regard them as being backward or even defective in their average mental endowment. The verbal responses are often quite inadequate, grossly diminished or even absent; and yet their poor scholastic achievements are not entirely attributable to an inborn lack of intelligence but, in fact, to an impaired functioning of one or more of their specific sensory organs. Inaptitude in reading, writing or numerical problems also occur as a result of these sensory handicaps. Cases of visual aphasia represent one of the most serious examples of non-readers; the milder aphasias, however, usually yield to appropriate remedial instruction. There are other physical causes, of course, that may mask or distort a child's native capacity wholly or in part. Impairment in hearing leading to deafness, motor aphasia, paralysis of the speech muscles or the inability to speak following social and cultural isolation in early childhood may considerably contribute to a condition resembling that of true mental deficiency. Moreover, there are children with ocular manifestations leading to varying degrees of visual impairment such as congenital cataract, gross errors of refraction and specific eye diseases. Retrolental fibroplasia, for instance, is one which is apt to develop in prematurely born babies who are exposed to excessive amounts of oxygen for an undue length of time, finally ending in blindness. This condition, of course, is preventable in many instances and is not attributable to genetic factors as originally thought. There are, however, some hereditary familial conditions associated with mental retardation which are complicated by morbid disorders such as optic atrophy, and retinal and other degenerative processes.

If sensorily handicapped individuals are neglected for a prolonged period of time, particularly during their most receptive, formative years of pre-adolescence, permanent signs of a functional mental deficit may ensue. The educational programmes in residential schools for the blind and deaf have demonstrated that the majority of such children, if given specific aids and suitable training, possess an inborn potential which assists them to develop quite adequately, often reaching satisfactory levels of educational and social achievement. In rare instances they may even reach the brilliancy of Helen Keller, Kaspar Hauser or Laura Bridgman.

Another factor in the causation of pseudo retardation is the influence of the cultural environment on the intellect.

A total lack of external stimulating conditions aggravated by a complete absence of human and educational influences, predisposes some individuals to intellectual stagnation of varying degrees, a coarseness of behaviour and even adoption of animal mannerisms. The physical and cultural isolation of human beings has occurred accidentally from time to time, and persons found under such circumstances have been referred to as "wild" or as being mentally backward. Owing to a lack of adequate intellectual stimulation, the higher thought processes have never had any opportunity or outlet of unfolding and thus their potentialities remained latent or undeveloped.

Such social isolation occurring during the first five years of life or so is held by many authorities to be the responsible cause of inability to learn to speak, or of poor speech habits. The child's mind at that age is most impressionable and plastic regarding its developments of thought associations and verbal expressions. Any neglect of cultural and training contacts during the pre-school period tends to imperil the child's chances to develop higher mental processes, to cerebrate on abstract matter and to conduct himself like a normally developed well-balanced person.

There is a school of psychologists who, having evolved a rather rigid concept, attribute supreme importance to the stimulating effects of cultural and social contacts upon the child's intellectual growth in subsequent years. This environmentalist school is of the opinion "that there is no escape from the fact that the intelligence quotients of children possess possibilities over a large area of the I.Q. range from genius to feeble-mindedness. Children can and do change in test I.Q. from average to genius and from average to feeblemindedness if they are under the continued influence of a stimulating environment or not".

According to this isolationist concept, true mental retardation results from any form of isolation if the latter cuts the person off in early childhood from any form of social, cultural or educational influences for a considerable period of time.

It is true that environmental factors do play an important role in the attainment of tested intelligence. This point has been particularly well brought out by the work of Goldfarb in 1940. In this he compared two classes of children; those who had spent the first few years of their lives in orphanages and those coming from a similar socio-economic background but who were brought up in foster homes. In later years the orphanage group that had excellent material care but little individual attention and who were emotionally starved, achieved an average I.Q. of 69, while the foster home children who had the opportunity of understanding, care and sympathetic interrelationship to individuals even in poorer economic circumstances, achieved an average I.Q. of 96. Therefore, it would appear that educational deprivation in early childhood does not affect children intellectually as seriously as deprivation of love and affection. It is the latter which apparently leads to a masked form of mental deficiency which in some cases remains irreparable and irreversible. No institution for the educationally retarded can ever hope to replace the warmth, friendliness and individual kindness of a motherly person even if she were poor or illiterate or both.

The consequences of maternal deprivation after the age of six months become progressively more serious and finally unalterable in some instances. As Spitz has noted, at two months the infant can recognize the mother's image and between the age of six to eight months he can distinguish the mother from strangers. If the separation of the maternal figure occurs under three months, the developmental quotient (D.Q.) would gain on the average 25 points after reunion with her; if the separation be somewhat longer, it would raise the D.Q. by some 13 points after reunion, and if the separation be still longer, rather less points on her return. Should the emotional isolation exceed five months, the regression would continue still further and become a progressively irreversible process.

A more serious finding, however, points to these children's inability or poor response to simple motor and verbal skills such as walking, running, and talking. Of twenty-one institutionalized children aged four years, Spitz noted that five could not walk at all; sixteen walked by holding onto furniture; twelve could not eat alone with a spoon; twenty could not dress themselves, only one could; six were not toilet trained at all, six children were unable to talk, five had a vocabulary of two words, one of a dozen words and only one actually spoke short sentences. All this tends to show and emphasize the fact that these children functioned for all practical purposes on a pseudoretarded level. The only child who was able to speak, walk, dress himself, eat alone and who was toilet trained, was a little boy of great beauty. He just looked like an angel and no person entering the nursery of the foundling home failed to stop at the child's bed and talk and play with him. This little boy obtained more attention, love, care and personal supervision by the nurses, doctors, and visitors than all the other infants put together. The remaining babies were reared by a nurse who did not have sufficient time to do anything more than hurriedly feed ten or twelve children who were in her charge. There was no opportunity of fondling them or of establishing individual contacts. It was further observed that the babies' facial expression became more rigid, frozen and immobile as the time of the emotional deprivation progressed and they gradually began to acquire a far-away and dazed look in their eyes. It would therefore appear that a period of three months separation of maternal attention causes the infant's D.Q. to regress on an average of 12½ points, from three to four months some 14 points and over 5 months about 25 points. The child separated from his maternal figure and thus emotionally isolated, loses gradually his previously acquired skills. In addition it has been noted that after a further period of separation from his familiar maternal image, he slowly gives up standing upright, walking and even sitting in his chair. He just lies down ready to fade away. His emotions become blunted and he ceases functioning on his developmental achievement level. The loss of his mental faculties still further leads to a lowering of his intelligence quotient and many of his former skills and abilities can never be regained. If the maternal separation extends beyond 5 months as above stated, the child becomes starved in his affective tone. This in its wake has a harmful effect on his mental and physical development and not infrequently leads to his premature death. An example of such extreme emotional deprivation which results often in an increased mortality rate, was observed in a Child Care Home on Randall's Island, New York, where of 366 motherless infants who were placed there for institutional care, only twelve were still alive a year later. In other words, there had been an infant death rate of 96·7 per cent. (Teagarden). Additional studies revealed that foundlings who were taken there under 6 months of age, had a very small chance of survival. These findings were established towards the end of the last century and conform more or less with the observations recently made by various investigators.

Infants need a personal, affectionate approach as well as individual care if they are to survive as physically and emotionally normal and intellectually balanced individuals. They continually need the fondling care, handling and love of their maternal figure. This personal attention is far superior and preferable to the best institutional nursing care and habit training if the child's mental development, physical growth and happiness are to be assured.

Goldfarb noted that institutionalized children show what he called "a definite level of conceptualization, a difficulty in organizing a variety of stimuli, an inability to think abstractly or to make generalizations in their learning ability". He further believed that the differential diagnosis between children who have suffered early affective deprivation through living in an institution from those who had been traumatized by early rejection, can be made. The differences, he thought, were primarily due to the institutionalized child's inability to identify himself with a particular loving adult, whilst the rejected child has been able to relate but negatively to a person. In the former there is a lack of positive relationship whilst in the latter a relationship already exists though only negatively. He believed that the early identification is important for the development of the child's psychic structure which provides the motivation for normal maturation and differentiation of personality.

With regard to the question of behavioural isolation it does not suffice to assume that isolation is the primary factor in the etiology of retardation. In fact, it is not the isolation of the individual which produces the mental handicap but rather his lack of imaginative response and action in relation to the surrounding, static isolation. He is unable to escape from the latter owing to his poor native endowment and lack of inborn drive. In 1913, Schlesinger expressed the view that an impoverished, unhygienic milieu largely contributes to pseudo-retardation and that harmful exogenous conditions are more frequently responsible for the exacerbation of a masked state of backwardness than for the production of mental deficiency itself. Laquer and von Dühring stated that primitive, unhealthy living conditions such as poverty, squalor and decay of family life are important contributory factors in the causation of intellectual impairment. As far as the domestic conditions of any one generation are concerned, it goes without saying that good living habits have an increasingly good effect while bad customs a correspondingly bad influence on the child's mental and physical growth. Children of parents who are in poor health and surrounded by bad hygienic living conditions are tempted to overlook essential measures for their children's own welfare. The well-established bad habits and neglect in early childhood are not likely to change because they have come to assume the responsibility of parenthood. Their children in turn may again experience similar unhygienic conditions handed down by their parents thus perpetuating conditions conducive to an unsatisfactory socio-economic and cultural environment. A poor social inheritance, if we may call it by that name, is therefore the end result of multiple factors such as malnutrition, and a lack of adequate moral, religious and educational background accompanied by a low standard of physical and mental hygiene. The writer is of the opinion that the existence of unsatisfactory, economically poor domestic conditions of the retarded is mainly attributable to the unwise management of their finances, to their poor scholastic opportunities, to their lack of robust health, and to their intellectual insufficiency. The writer further holds that lack of common sense, drive and emotional stability are more potent factors responsible for the low economic standard of the mentally defective than solely their educational subnormality. As a consequence one may be justified in saying that the retarded person's lower rate of income and social standing are primarily due to his limited emotional control, and lack of basic common sense and education rather than to his social inheritance of subcultural home conditions.

Another hypothesis is that the basis of individual sociality is social interaction. This interaction becomes effective if the individual can look upon himself as an object or can become aware in advance of the reactions of his environment. This hypothesis makes an attempt to apply sociological concepts to the etiology of mental deficiency. Such sociogenic and environmental concepts are becoming increasingly untenable, if they are viewed as the primary basic factors in the causation of true mental handicap. The writer for one is unable to identify himself with the isolationist hypothesis which regards isolation as the principle cause of mental retardation.

This school's dogmatic conclusions remind the observant reader of the concepts expressed by the Behaviourist School of Watson. "It is doubtful" stated Wallin, "if any mentally defective child of the endogenous group has ever been restored to partial or complete possession of an average mentality merely by the methods of training and education commensurate with his environmental pattern of culture." To attribute an individual's inborn intellectual deficit primarily to his inadequate surroundings or to his parents' inferior social and occupational living standards, is a view which, in the writer's judgment, is quite untenable and ludicrous. With this deduction in mind, the "Milieu Theory" is bound to fail, for it attempts to explain primary mental deficiency solely as a consequence of social isolation. With regard to the preponderantly inferior occupational standing of the progenitors of retarded children, their wretched environmental conditions tend to show significantly that the crucial point in question, the milieu—is not the cause of the mental handicap but the result of the parents' intellectual insufficiency.

Factors arising in the mind play an important role in the aetiology of the pseudo-retardation syndrome. Many school children found to be emotionally unstable give the impression of being defective merely because of unfavourable, or unattractive teaching methods. A child from a foreign country or from a small rural community, for instance, has greater difficulties in adjusting than a child born in his own country. A pupil unaccustomed to such a socio-cultural situation with its new lingual and educational patterns, may present a superficial likeness to that of a backward person. This particularly applies to children coming from groups ethnologically different from those in their new environment (bucoliphrenia). These deep-seated psycho-emotional tensions and frustrations complicated by personality and temperamental disturbances seriously interfere with the normal processes of intellectual maturation. If such neurotic children be neglected for a long period of time or are not given adequate professional advice, reassurance and counselling, their native mental endowment and aptitudes may remain stationary or under-developed. There is a possibility that they will deteriorate intellectually at such an alarming pace that they would conduct themselves like persons with limited or even subnormal mental capacity. Finally, they would show signs of functioning on a mental defective level possibly to an extreme degree.

A relative problem is the tendency of the emotionally unstable, nervous and apprehensive child to appear at a disadvantage in the testing situation. In some individuals such temperamental inconsistencies and emotional upheavals seem to cause little or no lowering of intellectual functions. In others, however, the effect may be appreciable and of considerable magnitude. The problem of rapport and of putting the testee at ease is a vital and predominant one. The question of the effect of fatigue upon the test performance is, of course, also an important one, depending on personality, temperamental and psychological factors such as motivation. Some children become so emotionally tense and apprehensive in the presence of an impersonal and unduly strict examiner that they may obtain a rating equivalent to that of a slow-learning child. This same pupil, however, when put at ease by a sympathetically oriented and experienced psychologist, may obtain a score of a dull normal or even of an average normal individual. The testee's mental functioning on a pseudodefective level may be nothing more than the result of the examiner's inexperience, abruptness or impatience.

Other factors arising in the psyche may also contribute to a considerable extent to pseudoretardation. Speech inhibition, psychic mutism, frequent attacks of petit mal, pyknolepsic lapses or even subclinical episodes of paroxysmal cerebral dysrhythmia, often arouse suspicion of mental backwardness on the part of the educator, psychologist, psychiatrist or pediatrician. In such psychogenically disturbed, emotionally unstable children, a programme should be planned which, in addition to improving their academic, manipulative and vocational skills, should aim at diminishing tension, temperamental instability and thought blockage, and help to improve work and study habits, self-confidence and social maturity.

Quite apart from the organically caused visual, auditory and neuro-orthopaedic defects, which are apt to impair seriously the child's intellectual functioning in addition to the severe dysrhythmic disturbances above described, there are also certain psychotic disorders which seriously interfere with the child's ability to respond normally and thus simulate an intellectual insufficiency.

At a meeting held by the International Institute of Child Psychiatry at Toronto, in August 1954, the following observations were made: "Until a few years ago of most cases of child psychosis or borderline ego disturbances diagnosed as mental deficiency in a routine manner, the majority probably found their way into institutions for the mentally defective, a fate which in future, it is hoped, will be avoided as far as it is humanly possible. It would be interesting to survey the populations of Training Schools, for it is probable that a careful investigation would lead to the discovery of many individuals falling within this pseudo-mental deficiency class. It is therefore well to emphasize that considerable caution be experienced by pediatricians, psychiatrists and psychologists with regard to a correct differential diagnosis of mental deficiency in a young child under three years of age lest iatrogenic factors add to the burdens imposed by nature."

The suggestion that emerges from the above observations and discussion is to investigate not only the full history of the child's development and past behaviour but also to be equally careful about the records dealing with the developmental data and difficulties arising from the child's relationship with his parents, particularly with regard to behavioural disturbances that antedate the child's signs of mental subnormality.

In considering the psychotic states in comparatively young children, while relatively infrequent, they occur, although quite a considerable number of them remain undiagnosed.

About four decades ago or so, childhood psychoses were considered practically non-existent, but thanks to the monumental contributions to psychiatry by the Bleulers and the introduction of reliable diagnostic criteria, it has been made possible to detect a sizeable number of psychotic responses in various aspects of their behaviour. Those with an I.Q. of less than 30, showing a psychosis resembling that of infantile schizophrenia, were described as featuring a clinical picture of "Larval Psychosis of Idiocy" a term recently introduced by MacGillivray, being the equivalent of Earl's "Primitive Catatonic Psychosis of Idiocy".

Fortunately infantile psychotic states only comprise a small proportion of the total psychoses occurring in adults.

Although the diagnosis of childhood schizophrenia which many would prefer to call infantile autism, prepsychosis or atypical development, is being established more frequently nowadays, this mental aberration continues to be unfamiliar to the majority of the medical profession. This leads to the situation whereby psychiatrists seldom have the opportunity to examine and treat schizophrenic children in their early stages until some years after their psychological disintegration and stereotyped regressed behaviour have become obvious and manifest to all concerned. Until quite recently many of these children have been regarded mistakenly as mentally deficient and have been relegated to Colonies and State Schools for the Mentally Retarded. It is therefore imperative that doctors, nurses and other child care workers become more familiar with the early diagnostic symptoms of, and treatment possibilities for, the infantile forms of schizophrenia.

In addition to childhood schizophrenia, other types of psychoses are also observed during that development period. There are the congenital syphilitic psychoses, the behavioural deviations following encephalitis lethargica which may be complicated by educational retardation and paranoid delusions, the traumatic psychoses, infectious diseases associated with mental derangement, psychoses with epilepsy and other primary aberrational disorders. The psychoses, because of their grotesque, bizarre patterns of symptomatology, may superficially resemble the more severe grades of mental deficiency and the examiner ought to keep this fact always at the back of his mind. By and large one should try to be extremely cautious and guarded about making a definite and final diagnosis of a psychotic disorder in a child of less than one year without the consultation of a team of specialists comprising a child psychiatrist, neurologist, psychologist, pediatrician and the child's own doctor.

In most cases of child psychosis complicated by mental defect, a history of normal mental and physical development data is found. These psychosomatic functions, however, tend to merge into an almost imperceptibly slow and increasingly abnormal pattern which culminates in a partial or complete loss of most of the acquired verbal and physical accomplishments.

The relationship of the infantile psychoses to oligophrenia has been discussed in some detail by Yerbury and others. Schilder pointed out, supporting A. F. Tredgold's observation, that a combination of a schizophrenic reaction state superimposed upon that of mental deficiency is not common and he further expresses the view that the syndrome resembling schizophrenia is at least in some cases the result of organic brain pathology. Space-occupying tumours involving the pre-frontal lobes, the pre-senile forms of dementia, e.g. Pick's disease, intracranial vascular lesions and the Gerstmann syndrome of dementia paralytica, predispose to the development of a mental syndrome simulating that of catatonia. A catatonic reaction state should also be thought of in those children whose frontal brain areas have been injured or singled out by hereditary, familial diseases.

There are authorities who are of the opinion that the defectives possess a certain resistance to developing mental disorders. Some maintain that only very few among them actually become of unsound mind. They believe that their relatively simpler nervous system renders them less sensitive to disturbing influences than is the mentally normal individual. Others again hold that the retarded, because of their inborn instability and frequently occurring developmental defects of the central nervous system, lose their mental balance readily under conditions of social stress and strain, economic competition and drugs. Consonant with this view, the writer is inclined to think that the retarded are more prone to neuroses than intellectually normal persons; however, they appear to be less frequently subject to states of psychoses.

Recent estimates of Neuer claim that at least twenty per cent. of persons who function on a high-grade level of mental deficiency manifest signs of a minor or major psychoneurosis or psychosis. The writer considers this figure too high. In his view, the incidence of psychosis in a Training School population ranges from one to two per cent., and that of neurosis approximately from four to six per cent., making a total of from five to eight per cent. for neurotics and psychotics combined. This figure is not appreciably at variance with the incidence of neurosis and psychosis among intellectually normal persons.

The classical condition of manic-depressive psychosis as presented in its clinical form of cyclothymia is infrequently met with among the mentally defective. The author is of the opinion, however, that retarded persons are never truly melancholic or manic as observed in the intellectually normal, they rarely display the clinical picture of the typical circular psychoses. Manic-depressive insanity is primarily a mental aberration affecting essentially highly integrated, intelligent individuals, which cannot, however, be said of our group of mentally subnormal persons.

The second most important group of mental disorders, the schizophrenic reaction states, are basically conditions of psychological disintegration and splitting of the personality as an entity. From this it would appear that the mental defective might be particularly prone to this schizophrenic form of behaviour deviation because of their poor quality of personality integration. The mental defects, as is well recognized, are essentially unintegrated in their psychological structure and the schizophrenic disorder is basically a disintegrating condition. It is therefore to be expected that fragments and atypical clinical combinations of sign and symptoms are found not infrequently among the defectives. In fact, the process of psychological fragmentation and disintegration makes the clinical differential diagnosis so complex and disconnected between intellectually defective and educationally normal psychotics that there is a tendency to regard the diverse scraps and dismembered pieces of behavioural deviation as being primarily schizophrenic in aetiology. The writer is convinced that this cannot be so, unless we are dealing with truly paranoid or hebephrenic forms of schizophrenia observed in high-grade mental defectives and in persons of borderline, or normal mental capacity.

The absence or presence of a wide scatter may in many instances help diagnose between familial forms of mental deficiency and psychotic states such as schizophrenia.

The familial defective conversely does not manifest a wide range of responses on a psychological test. Among those whose responses do scatter through several years they may be either schizophrenic or organically affected defects.

In cases where a psychotic condition, organic nervous disease or epilepsy, is associated with a state of mental deficiency, the clinical diagnosis should be primarily based upon the developmental data, family history, educational record, neurological examination and mental condition of the patient. Séguin in 1866 made the first observation that the psychoses are not infrequently connected with low-grade mental defect. He divided the psychotic disorders into the hyperkinetic and hypokinetic forms. Griesinger, in 1867, drew up a similar table classifying the aberrations as either apathetic or excitable. He regarded the signs of destructiveness, restlessness, aggressive conduct and fretfulness as expressions of a "pure mania".

MacGillivray, in his monograph on "The Larval Psychosis of Idiots", fully reviewed the early literature of mental deficiency complicated by psychosis, and stated "that A. F. Tredgold in 1908 considered the sudden violent brain-storms of idiots to be indicative of true insanity", and Barr claimed that insanity in the mental defective could be diagnosed as early as the first year of life; Kraepelin retained the traditional dichotomy which he renamed erethic and apathetic and described primitive reactions such as screaming attacks, rage, stupor, reactive depressions and manic-like attacks. Many of the mentally defective belong to the pathological group (idiots and imbeciles) who three or four decades ago were described as showing overt signs of a psychosis associated with gross hyperactivity. They were probably brain-injured individuals with a cerebral pathology and not true types of mental defect aggravated by a state of psychosis.

Hurd's belief was that attacks occurring in the severely defective should not be considered as episodes of insanity, and Benda observed that 56 per cent. of syphilitic idiots in his series demonstrated definite psychotic signs associated with specific neurological conditions such as tabes dorsalis or dementia paralytica. Penrose in an extensive study of 1,280 cases of mental defect noted that some showed quite definite psychotic signs, such as mannerism, negativism, catatonia and stereotyped behaviour. The writer has detected 12 defectives amongst a resident population of 1,853 mentally retarded persons exhibiting clinical signs of catatonia, resistiveness, and negativism, and in seven mid-grade defectives additional signs such as hallucinatory experiences, waxy flexibility, catalepsy, echopraxia and echolalia. Emotional disturbances and thought blockage were also observed.

Earl, in a series of 135 idiots, was able to define a sub-group of 38 defectives, as showing a syndrome consisting of catatonia, emotional dissociation, inaccessibility, deterioration in personal and toilet habits and indistinct articulation. He named this syndrome "Catatonic Psychosis of Idiocy", believing these psychoses "to be a form of schizophrenia played out at a motor level instead of the symbolic". Catalepsy alone may often occur as a complication in mongols and tuberous sclerotics. Rollin, quoted by Wallin, classified, 23·3 per cent. of psychotic mongols as showing a primitive type of schizophrenia.

There is, in fact, an appreciable number of children who have been evaluated as manifesting a state of pseudoretardation. The writer has observed many a patient who has never emerged from a chronic condition of psychotic illness and who throughout his life could not be psychometrically differentiated from a severely retarded person of primary or secondary aetiology. Under such circumstances one may be justified in doubting as to whether individuals belonging to the dilapidated, chronic group of psychotics should be classified as mentally deficient because of their inability to respond adequately to psychometric examinations. Before including these mentally reduced psychotics whose social and intellectual withdrawal symptoms commenced during their early childhood or adolescence, into the group of mentally defective persons, one ought to emphasize their resultant mental retardation differing in aetiology from all those customarily referred to as mental defectives, be they of endogenous or exogenous origin. Following many years of unrelieved, chronic psychotic illness, some patients degenerate from a state of chronic dementia to a condition of secondary oligophrenia. The speed of this intellectual reduction depends more or less on their constitutional and mental make-up, heredity and environment. Hecker, describing hebephrenia in 1871, stressed its appearance in connection with adolescence and emphasized the frequent and rapid deterioration of this psychosis into a condition of severe mental loss. A mental condition commencing insidiously or rapidly in late childhood years or adolescence is usually one of serious import for it often has a tendency to lapse into progressive levels of mental dullness or varying degrees of mental retardation. Such persons finally sink into a state of complete obliviousness to the environment, resembling a state of Wahnsinn-retardation. (Wahnsinn—insanity.)

The incidence of mental retardation in psychiatric hospitals fluctuates from five to ten per cent. of the total residential population.

Let us now proceed with the description of several important clinical syndromes which may be easily confused with mental subnormality.

In 1943, Kanner described a syndrome beginning in early infancy which is characterized by extreme introverted behaviour, withdrawal from contact with persons and features of dereism. To this syndrome he gave the name of early infantile autism. At first he regarded it as a distinct clinical entity but now in unison with the majority of child psychiatrists, he believes it to be identical with a schizophrenic state of the earliest possible onset. This infantile form of psychosis is sometimes referred to as prepsychosis or childhood psychosis.

During the early autistic phase of the schizophrenic illness, the children begin to reveal an emotional blockage, an inability to establish an effective relationship with their mother, revealing a permanent lack of emotional responsiveness, disturbances of associative thought, a tendency to divorce themselves from real existence and a preference for day dreaming. The parent begins to fear that her child is mentally defective because he does not talk on reaching his third or fourth year or because of his inability to relate to people adequately. The child's dereistic behaviour, extreme introversion and limited responsiveness give the impression that he functions on a mentally retarded level although it is subsequently found that he is of average or even superior intelligence. Those, for instance, who possess power of speech use an extensive vocabulary. Their facial expression is often remarkably bright and intelligent in spite of their continued divorce from interpersonal relationship. They are self-occupied and demonstrate peculiarities of memory, language and motor performance.

As the child grows older, however, he gradually begins to reveal the true nature of his mental illness. It becomes daily more clearly manifest that the clinical picture of his aberration is one of childhood schizophrenia as observed in adults in its fully developed state. Many of these children continue to function in an emotional vacuum although there are a few who attain normal intelligence and show some intermittent attention to persons and activities in their surroundings. However, in the adolescent, delusions and hallucinations become insidiously established. Some begin to display moroseness, moodiness, obsessive rumination and sudden unpremeditated outbursts of aggressive conduct. They show acute features such as grotesque, peculiar postures, an impaired ability to concentrate with an accompanying reduction in the standard of their scholastic work. They begin to sleep poorly, show a loss in appetite, mumble to themselves in response to imaginary voices, become inaccessible and dereistic. A period of lucidity at times ensues followed by a further exacerbation leaving them increasingly disorganized mentally and also with a changed personality. For practical purposes such a person is educationally subnormal and in the majority of cases ineducable owing to his progressive dissociative psychosis. The existence of schizophrenia in later infancy or childhood should be differentiated from mental defect, organic cerebral pathology, severe neurotic disorders, and behavioural disturbances. The latter result from deprivation of love and warmth on the part of parents who are "refrigerated and who cannot defrost" (Bender).

There are many retarded children, who on developing a psychosis in later life, demonstrate a pronounced delay in physical and mental development. We therefore require adequate and reliable methods in order to differentiate dependably and satisfactorily the mental defective from the psychotic. The very basis for an accurate diagnosis still rests on a detailed and reliable case history. The defective's inability to learn abstract subject matter and to adjust to normal situations in an emotionally mature manner are two of his most outstanding shortcomings. The psychotic, however, in early life, appears to be quite rational at first, emotionally stable and intellectually normal until some serious obstacle arises. He then begins to show signs of mood swings or a loss of contact with reality and finally loses all interest in his playmates, toys and environment. A sudden failure in his studies, a cyclothymic lability, catatonia, negativism, waxy flexibility, mutism and catalepsy are frequent prominent signs observed in adolescent psychotics.

The differential diagnosis of the more complicated cases of pseudo-mental deficiency requires the services of experts well trained and experienced in psychiatry, neurology, medicine, clinical psychology, mental deficiency and in methods of special education. The combined clinical result of the examinations and the scholastic evaluation should preferably be supported by additional psychometric, electroencephalographic and other diagnostic aids in order to arrive at a scientifically reliable diagnosis. Owing to an extensive overlap of psychoneurotic symptoms and a large variety of psychotic reaction states observed in both high- and low-grade mental defectives, it may be proper to describe these groups of patients as belonging to the category of mental retardates of the unstable type, namely the mentally subnormal with an engrafted insanity of "Pfropfpsychotics". The latter can be subdivided into the Pfropf-Schizophrenics and the Pfropf-Cyclothymics.

In 1896, Kraepelin defined the term Pfropf-Hebephrenia by explaining that in such cases "dementia praecox was in a certain manner grafted upon weakmindedness which had existed from childhood without local phenomena". The term Pfropf-Hebephrenia was then changed to Pfropf-Schizophrenia by Eugene Bleuler in 1911, whose suggestion it was to supplant the term dementia praecox by that of schizophrenia.

In an article written by Myerson dealing with mental deficiency and schizophrenia, he stated that "now and then one sees a feeble-minded patient suddenly enter into a deluded, hallucinated state, ending with marked mental deterioration which in a previously normal mentality would be called schizophrenia. On the whole it seems to be unlikely that there exists any biological connection between the two sets of conditions namely schizophrenia and feeblemindedness. The schizophrenic's disintegration of mental processes seems entirely different in nature from that of the inborn limited capacity of the feebleminded."

The etiological relationship existing between the hereditary forms of mental retardation and the functional psychoses has been fully discussed by Wallin in his book entitled *Children with Mental and Physical Handicaps* (1950). Some of the conflicting views universally held on their causal relationship are quoted as follows: "Both conditions have a common aetiology originating from the same genotype; both conditions are the outcome of a genetically tainted vulnerable brain sensitive to pre-, para- or post-natal traumas; there appears to be a compensatory mechanism at work between the two entities which tends to influence these mental conditions in a modifying manner. Mental deficiency changes the character of the schizophrenic illness and alternately the latter reinforces the state of mental retardation; the occasional concurrence is purely accidental. Others, however, maintain that there are no aetiological connections between these two conditions."

There are several other clinical syndromes simulating mental deficiency which, however, reveal a variety of different causative factors; amongst these we have, for instance, Heller's disease, lead encephalopathy and dementia praecocissima. These clinical entities are not infrequently responsible for so rapid a deterioration of a mental function that a child is quite often diagnosed as suffering from a galloping form of mental deficiency.

Heller's syndrome or dementia infantilis, is a progressive illness. Such children develop normally or precociously without any previous evidence of neurological, psychotic or intellectual subnormal history until they reach their second, third or fourth year of life. Before the onset of behavioural disturbances such as hyperactivity, restlessness, emotional instability and aggressive destructive conduct, some children appear to be quite normal and active. Others, however, exhibit fleeting hallucinations and delusions, mumble to themselves, become forgetful, inattentive, careless in their appearance and echolalic. They regress into mute behaviour and finally lose complete command of their power of speech. Language and emotional maturation begin to lag a little earlier. Neuronal degeneration involves the cerebral cortex, cerebellum and spinal cord and Corberi found in four biopsies "diffuse, acute lipoid degeneration of the ganglion cells" suggesting that their condition represents an inborn error of lipid metabolism such as is observed in the cerebro-retinal degenerations and Niemann-Pick's disease. Kuf described the last-mentioned condition as "Amaurotic Idiocy without Amaurosis". Schilder held that "dementia infantilis had nothing to do with oligophrenia but was an organic degenerative process culminating in pseudo-retardation". Kennedy and others reported cases in which progressive intellectual deterioration was directly referable to infantile pertussis, the children finally becoming defective in spite of the retention of an alert, attractive facial expression.

In children suffering from convulsions of more or less sudden onset and in whom some form of mental retardation has also been suspected, lead encephalopathy should be always thought of and excluded as an aetiological factor. In cases of lead poisoning in childhood the intellectual growth of the individual is in many ways seriously hampered. The death rate of children under five years of age is pretty high and the post-saturnal effects result in many cases in a certain degree of intellectual backwardness which often ends up in mental retardation. Lord made a follow-up study of twenty children who had received hospital treatment for lead encephalopathy in the pre-school age. All of these children, with the possible exception of two, made a very poor adjustment both emotionally and scholastically in the ensuing years. For this reason it is essential to see that prophylactic and legislative measures be taken promptly to prevent children from ingesting or inhaling lead particles from sources which may contain lead. Post-saturnal backwardness and mental deficiency are the result of neglect, lack of supervision and preventative care. It is of importance for the clinician to obtain a thorough history in all cases where very young children show intellectual deterioration so as to exclude the possibility of chronic lead poisoning, the source of which may otherwise remain undiscovered.

In 1905, De Sanctis described a condition of pseudo-mental retardation which he named dementia praecocissima; it is now considered to include a group of mental disorders of functional or organic etiology, which commence about the fourth year of life. The children exhibit some emotional blunting, gross intellectual loss and catatonic behaviour. These symptoms appear to point to a primary involvement of the prefrontal and frontal areas.

SUMMARY

In this paper an attempt has been made to discuss possible conditions and clinical syndromes resembling true mental deficiency; it has been shown, however, that some of the neuroses, psychoses and diseases of the central nervous system simulate various types of mental retardation. Early environmental isolation and affective deprivation have been described as possible aetiological factors in the development of secondary types of oligophrenia. Great skill, patience and experience are needed by the clinician in order to arrive at a reliable evaluation of each individual case. The simultaneous existence or sequence of a psychosis superimposed upon a state of mental deficiency in the same individual has also been discussed at some length, and the world literature and views of different authorities reviewed.

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THE PHYSIOGNOMIC, PSYCHOMETRIC, BEHAVIORAL AND NEUROLOGICAL ASPECTS OF PHENYLKETONURIA

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PHENYLKETONURIA is an inborn metabolic disorder affecting the body's ability to hydroxylate phenylalanine to tyrosine.

Its incidence in the general population has been estimated at 0·004 per cent. and its institutional frequency at approximately 0·2 to 0·9 per cent.

It is generally held that phenylketonuria occurs usually in the Anglo-Saxon race and only to a lesser degree in the Semitic and Negro races. A recent study made on 1,853 patients at Caswell Training School reveals twelve phenylketonurics, putting the institutional frequency in this survey at 0·6 per cent.

The deficiency in tyrosine and the impaired formation of melanine leading to a sparse pigmentation of the iris, hair and skin, make one quite rightly conjecture that the hypo-pigmentation is due both to a quantitative reduction of melanine as well as to its frequent occurrence in the Anglo-Saxon race.

The physiognomy of phenylketonurics is fairly constant and of remarkable likeness in colour; they are distinguishable from the majority of mental defective patients by their characteristic light-coloured hair, iris and skin. In many of them these structures are less pigmented than in their parents and some have quite attractive features and pretty complexions.

Of the twelve phenylketonurics examined, all are blond, eleven blue-eyed and only one brown-eyed. Two have a very pale skin, six a pale and four a normally pigmented integument. Dermatographia and eczema have been noted in the histories of two of these children (Table I, Cases 2 and 5) and one case of photophobia in a girl (Case 8).

TABLE I

Pigmentary Characteristics Shown in Twelve Phenylketonurics

			Colour of			
Cases	Sex	Date of Birth	Iris	Hair	Skin	Special Features
1 (G.J.G.)	F	1.7.48	Brown	Blond	Normal	—
2 (J.C.O.)†	M	10.10.39	Blue	Blond	Very pale	Dermatographia. History of eczema.
3 (T.F.N.)	M	27.3.43	Blue	Blond	Pale	—
4 (V.B.)	F	12.6.19	Blue	Blond	Normal	—
5 (N.L.O.)†	F	26.1.41	Blue	Blond	Very pale	Dermatographia. History of eczema.
6 (N.D.B.)*	F	1.4.43	Blue	Blond	Pale	—
7 (C.D.B.)*	M	29.3.48	Blue	Blond	Pale	—
8 (M.K.P.)	F	8.4.48	Blue	Auburn	Pale	Photophobia.
9 (B.C.)	F	9.5.22	Blue	Blond	Normal	—
10 (M.E.S.)	F	16.12.30	Blue	Blond	Normal	—
11 (C.D.K.)	M	20.2.40	Blue	Blond	Pale	—
12 (R.M.B.)	M	25.10.30	Blue	Blond	Pale	—

* Siblings. † Siblings.

The phenylketonurics' behaviour and general conduct during the psychometric test evaluation with the psychologist have shown in eleven out of twelve cases gross hyperkinetic distractibility, lack of rapport, mischievousness, motor perseveration and echopraxia, and an extremely short-lived span of attention and interest in their surroundings. Stereotyped posturing, grimacing and mannerisms have likewise been observed in several cases.

On the whole their affective tone is somewhat blunted and their mood is in a state of constant flux, Their superficially cheerful, self-centred behaviour is apt to give way suddenly, and for no apparent reason, to short bursts of noisy restlessness, fidgetiness, aimless destructiveness and confusion; some are even subject to phases of enforced crying and laughter devoid of genuine emotional experience (Table II).

TABLE II

Behavioural Responses in a Group of Twelve Phenylketonurics During the Psychometric Test Interview

Cases	
1 (G.J.G.)	This is a fidgety, distractible, restless manneristic little girl who periodically whines. She shows an impaired awareness of her surroundings, has a short attention span and is emotionally unstable. She can, however, respond to simple commands, shows moderately purposeful movements and fluctuates between phases of interest and detachment in the psychometric test situation. Intermittently she ejects a fairly well articulated word or two. Grimacing and catatonic-like posturing have also been noted.
2 (J.C.O.)	He is a friendly boy who grins broadly when spoken to, showing little attentiveness and poor thought associations. He manifests pronounced motor and verbal perseveration, has hand tremors, is restless, overactive and capable of saying simple words with difficulty and slurred enunciation.
3 (T.F.N.)	This is a comparatively sociable, co-operative, mischievous and periodically restless boy. He is myoclonic, mute and of fixed countenance. When examined he becomes fidgety, gesticulatory with noticeable hand tremors, distractable and subject to pseudo-affective spells of laughter and grinning. He has a pronounced grasp reflex and needs prompting and prodding.
4 (V.B.)	She is an inattentive spastic woman taking little heed of the psychologist. She has choreiform-athetoid movements which follow a haphazard course, is restless, overactive, mute and poorly toilet controlled.
5 (N.L.O.)	This is an hyperactive, restless, tremulous, gesticulatory girl who grins and grimaces throughout the interview showing little comprehension. She is echopractic with verbal and motor perseveration. Her speech consists of a few indistinctly articulated words and her posture is one of moderate flexion resting her head on her chest.
6 (N.D.B.)	She is a happy but mute child who easily becomes excitable and explosive in temperament. Periods of hyperactivity are followed by those of apathetic, listless behaviour with total indifference to her surroundings. Her co-operation is only moderate.
7 (C.D.B.)	This is a lethargic and apathetic boy. At times, however, he is subject to phases of violent excitability associated with purposeless movements and aimless conduct, needing control. He is unco-operative and oblivious to his surroundings. His muteness increases still further the lack of rapport between the patient and the psychologist.
8 (M.K.P.)	She is a restless, noisy, negativistic, mute and totally unco-operative little girl, who is terribly frightened by female adults. Once, however, she leaves the examiner's room, she is a happy, sociable child although frequently destructive and mischievous.
9 (B.C.)	This is an apathetic, listless and lethargic woman who co-operates very poorly. Periodically she becomes overactive, excitable and restless for no apparent reason only to quiet down again after a few minutes or so. She is mute but able to respond to very simple commands and occasionally will smile fleetingly at the psychologist.

10 (M.E.S.) She is an apathetic, blind, mute and spastic woman. She infrequently emits inarticulate sounds, is wont to touch each object for identification, shows little co-operation and lives in a world of her own. At times she becomes restless pushing her fellow mates around the room, even hurting them. It is almost impossible to evaluate her psychometrically.

11 (C.D.K.) He is a poorly developed, mute boy who is wont to push all the testing materials off the table during the interview. He is indifferent as to where the objects fall and sits there with a vacant facial expression, He is restless, inattentive, resistive and unco-operative. He is fond, however, of holding a rubber doll in his arms, rocking himself vigorously in a rocking-chair.

12 (R.M.B.) He is an apathetic, mute, inattentive resistive boy of slow movements. He cannot talk but babbles to himself in an inane manner, bites and scratches his hands and arms and shows a lack of purposeful motions. He can, however, respond at times to very simple commands if they are repeated to him.

The most striking clinical finding, however, is the constant association of this metabolic disorder with a severe degree of intellectual deficit. All patients in the study group are mental defectives functioning on an extremely low level of cognitive efficiency.

Observations made by Jervis show that two-thirds of 161 phenylketonurics detected among twenty thousand three hundred patients in mental deficiency institutions in the United States, have been classified as idiots (66·7 per cent.) and one-third as imbeciles (33·3 per cent.).

The psychometric test data of the twelve phenylketonurics in our survey, manifest similar degrees of intellectual impairment. Nine of them (75 per cent.) fall within the range of severe mental deficiency with intelligence quotients (I.Q.s) varying from four to seventeen, while only three patients (25 per cent.) have I.Q.s from twenty to thirty-seven.

Repeated psychological examinations as described in Table III, show that

TABLE III

Rate of Change in Mental Ages of Patients with Phenylketonuria

Case	Date of Birth	Date Tested	Name of Test	I.Q.	C.A. in Months	Growth in C.A. in Months	M.A. in Months	Gain or Loss in M.A. in Months	Average Annual Rate of Change in Mental Age Expressed in Months	
									Gain in Months	Loss in Months
1	1.7.48	1955	St.Bi.L.	37	81	—	30	—	—	—
(G.J.G.)		1957	St.Bi.L.	35	111	30	39	+9	3·6	
2	10.10.39	1950	St.Bi.M.	20	128	—	25	—	—	—
(J.C.O.)		1957	St.Bi.L.	22	216	88	41	+16	2·2	
3	27.3.43	1952	Rome-Gesell	11	114	—	12	—	—	—
(T.F.N.)		1957	Cattell	11	175	61	19	+7	1·4	
4	12.6.19	1941	Kuhlmann	5	260	—	6	—	—	—
(V.B.)		1957	Kuhlmann	5	460	200	9	+3	1·8	
5	26.1.41	1953	St.Bi.L.	20	151	—	28	—	—	—
(N.L.O.)		1957	St.Bi.L.	16	201	51	31	+3	0·7	
6	1.4.43	1951	Cattell	6	96	—	7	—	—	—
(N.D.B.)		1957	Cattell	5	168	72	7	0	0	
7	29.3.48	1954	Kuhlmann	12	76	—	9	—	—	—
(C.D.B.)		1957	Kuhlmann	8	115	39	9	0	0	
8	8.4.48	1956	Kuhlmann	17	102	—	17	—	—	—
(M.K.P.)		1957	Kuhlmann	16	113	11	17	0	0	
9	9.5.22	1928	Rome-Gesell	26	77	—	20	—	—	—
(B.C.)		1957	Rome-Gesell	7	428	351	12	−8	—	0·2
10	16.12.30	1951	Rome-Gesell	6	333	—	12	—	—	—
(M.E.S.)		1957	Cattell	7	407	74	10	−2	—	0·3
11	20.2.40	1955	Kuhlmann	4	183	—	7	—	—	—
(C.D.K.)		1957	Kuhlmann	4	212	29	6	−1	—	0·3
12	25.10.30	1954	Vineland	7	290	—	20	—	—	—
(R.M.B.)		1957	Cattell	4	333	34	6	−14	—	4·9

The tests used throughout the years were:
The Revised Stanford-Binet Scale—Forms L and M.
The Infant Intelligence Scale—Cattell.
The Kuhlmann Test of Mental Development.
The Rome State School Adaptation of the Gesell Developmental Norms.

TABLE IV

Family History and Developmental Data

Case	Family History	History of Convulsions	Number of Siblings Excluding Patient	Birth Weight	Onset of Walking at	Onset of Talking at
1 (G.J.G.)	Mother mentally disturbed during pregnancy.	Fits began at one year; free from fits since 3rd year of life.	One	5 lb. 3 oz.	Two years.	A few words at four years.
2 (J.C.O.)	Paternal grand-father insane. Father blind in left eye.	Had fits during first few months of life.	Four (3 mentallv defective)	8 lb. 10 oz.	Five years.	Seven years.
3 (T.F.N.)	Maternal uncle had spina bifida. Maternal aunt diabetes.	Had convulsions shortly after birth.	Four (1 mentally defective, 1 crippled)	Not stated.	Not stated.	Mute.
4 (V.B.)	Paternal aunt is a mental defective and a psychotic.	Unknown.	Six (2 mentally defective)	Not known	Not known	Six years.
5 (N.L.O.)	Paternal grand-father is insane. Father is blind in left eye.	Had fits during the first few months of life.	Four (3 mentally defective)	6 lb. 0 oz.	Six years	Four years. Very poor speech.
6 (N.D.B.)	Two maternal uncles died in infancy following fits.	Fits commenced at three years. They continue to be of the major type.	Eight (2 mentally defective)	Not stated	Cannot walk	Mute.
7 (C.D.B.)	Same as above.	3 fits at 3 years; recent fit in spring of 1952, also 54 (4 years old) and now on anti-convulsive therapy.	Same as above.	6 lb. 1 oz.	Cannot walk	Mute.
8 (M.K.P.)	Father and mother as well as 1 brother mentally defective.	None.	One (mentally defective)	7 lb. 8 oz.	Two and a half years	Mute.
9 (B.C.)	Father is a diabetic; also obese and tuberculous.	Convulsions at birth.	Four (1 mentally defective)	Not known	Five years	Mute.
10 (M.E.S.)	Paternal grand-mother was tuberculous; 2 maternal uncles died as infants. Maternal aunts were obese and alcoholics.	Fits began at 1 year. Since his third year, free from fits.	Five (Two are dead and 1 is mentally defective)	9 lb. 0 oz.	Not known	Mute.
11 (C.O.K.)	Father is mentally defective. Two paternal cousins had crippled feet. Aunts, uncles and cousins died in infancy.	None.	Eight (1 mentally defective)	7 lb. 8 oz.	Cannot walk	Mute.
12 (R.M.B.)	One sibling died aged 8 months. Two siblings are mentally defective (1 ?P.K.).	Abnormally restless after birth.	Six (2 mentally defective)	Not stated	Three years	Mute.

TABLE V

Neurological Data in Twelve Phenylketonuric Patients

Case	Types of Involuntary Movement, Gait, etc.	Sex	Spasticity or Rigidity	Abnormal Findings in the Central Nervous System	Associated Hyperactive Movements	Clinical Diagnosis
1 (G.J.G.)	Jerky gait with brisk motions of arms and legs.	F	None.	Deep tendon reflexes within normal range. Babinski negative. No cerebellar signs.	Restlessness with stereotyped motions and grinning. Very active.	Uncomplicated type of phenylketonuria.
2 (J.C.O.)	Hand tremors. Gait normal.	M	None.	Deep tendon reflexes within normal range. Negative Babinski.	Overactive and restless.	Uncomplicated type of phenylketonuria.
3 (T.F.N.)	Pronounced myoclonic jerks. Parkinsonian mask. Slow hand tremors.	M	Some cog-wheel rigidity upper limbs.	Grasp-Reflex positive (Darwinian). Talipes planus with pithecoid stance. Tendon reflexes normal.	Rocking movements.	Striatal syndrome type of phenylketonuria.
4 (V.B.)	Athetoid-choreiform movements of hands, arms and legs.	F	Left side more spastic than right.	Triceps, biceps, knee and ankle jerks brisk; bilateral, positive Babinski. Diplegia.	Restless.	Cerebral palsy type of phenylketonuria (Chor Ath.).
5 (N.L.O.)	Fine tremors hands and fingers.	F	None.	Deep tendon reflexes a little brisker than normal.	Tremulous, restless.	Uncomplicated type of phenylketonuria.
6 (N.D.B.)	None.	F	None.	None.	Hyperactive.	Uncomplicated type of phenylketonuria.
7 (C.D.B.)	None.	M	None.	None.	Aimless motions; fidgity.	Uncomplicated type of phenylketonuria.
8 (M.K.P.)	None	F	None	Normal reflexes. Kyphoscoliosis.	In constant motion, overactive and destructive.	Uncomplicated type of phenylketonuria.
9 (B.C.)	No tremors.	F	None	Brisk upper and lower reflexes; negative Babinski.	Restless.	Uncomplicated type of phenylketonuria.
10 (M.E.S.)	Scissors gait.	F	Spasticity of all limbs.	Brisk tendon reflexes. Patellar (uni) and ankle clonus (bil) pos., blind in R. eye (cataract), nystagmus, *snout reflex*; cerebellar ataxia, paraplegia. Bil. Babinski.	Hyperactivity.	Cerebral palsy type (Little's Syndrome) of phenylketonuria.
11 (C.O.K.)	None.	M	None	None	Restless.	Uncomplicated type of phenylketonuria.
12 (R.M.B.)	None	M	None.	Normal reflexes.	Slow movements.	Uncomplicated type of phenylketonuria.

there has been a slow but clearly discernible growth in the mental age of five patients (Cases 1 to 5); no change, however, has been recorded in three patients (Cases 6 to 8), and a mental age loss in four (Cases 9 to 12). None of the above tested were ever placed on any specific therapeutic diet or medication.

It may therefore be assumed that a greater number of defective phenylketonurics would have achieved a higher mental age if they had been given the benefit of a phenylalanine-free preparation soon after birth, as the phenylalanine splitting enzymes derived from the mother's circulation and stored in the infant's liver become gradually depleted during the neonatal period leading to this condition.

The social and developmental data on Table IV show that only six patients are capable of walking unaided and that they have learned to walk between the ages of two years and six after a considerable delay and much practice. Their ability to articulate has been negligible and eight patients have remained without the power of speech. Four are capable of saying a few words in a rather indistinct, slurred and almost unintelligible manner.

Convulsions have been frequently mentioned in the histories of our study group. Four (33·3 per cent.) have been subject to seizures from birth to one year, and four (33·3 per cent.) have had convulsions commencing at one year of age and upwards. Two phenylketonurics continue to have epileptic seizures of the major variety (Table IV, Case 6, 7). Their weights at birth varied from five pounds three ounces to nine pounds.

Labour has been described in five cases as being particularly difficult and prolonged, requiring the aid of instruments and a general anaesthetic.

Of twelve neurologically examined phenylketonurics two have revealed a clinical diagnosis of cerebral palsy and one a striatal syndrome (Table V). The cerebral palsy group consisting of spastic and choreo-athetoid types, comprises one patient with diplegia (Case 4), and one with paraplegia and pronounced scissors gait (Case 10). The remaining eight patients belong clinically to the uncomplicated type of phenylketonuria (Table V; Cases 1, 2, 5, 6, 7, 8, 9, 11 and 12).

Choreiform movements have been observed in one patient (Table V, Case 4), athetosis in one (Case 4), hand tremors in three (Cases 2, 3, 5) and myoclonic jerks in one patient (Case 3). The extensor type of plantar response (Babinski) has been recorded in two (Cases 4, 10), the ankle clonus in one (Case 10), the patellar clonus in one (Case 10) and increased or exaggerated deep tendon reflexes either uni- or bilaterally in four patients (Cases 4, 5, 9, 10). In one the grasp reflex (Case 3) has been elicited and in Case 10 the snout reflex.

Skeletal deformities have been noted in two patients, one showing marked bilateral talipes planus associated with pithecoid stance and striatal disease (Case 3), and another kyphoscoliosis (Case 8).

Further investigation has also brought to light the incidence of fifteen infant and sibling deaths in seven families, alcoholism in three, psychosis in four, mental deficiency in eight, tuberculosis in two, gross obesity in two and diabetes in three (see Table IV).

SUMMARY

Twelve cases of phenylketonuria have been described showing characteristic pigmentary deficiencies in their ectodermal structures.

Hyperkinetic, distractible behavioural disturbances have been outlined.

The growth in chronological age of each patient has been expressed in months and the corresponding gain or loss of mental age has also been calculated in months.

In addition, the average annual rate of change in mental age expressed in months has also been tabulated. Five patients have shown a positive gain, three no change and four a loss in

their intellectual capacities. None of the patients have received any specific therapeutic dietary preparation.

The social and developmental histories of the study group have been given, showing a pronounced lag in their maturational growth and a frequent incidence of psychological and clinico-pathological findings in their family histories.

The neurological data as outlined are indicative of an involvement of the pyramidal, striatal and cerebellar systems, describing two patients with signs of cerebral palsy and one with striatal disease.

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THE CARE AND TREATMENT OF PSYCHOPATHIC OFFENDERS IN NORWAY, SWEDEN, AND DENMARK

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In 1956 the Henderson Research Trust awarded the writer a scholarship which enabled him to spend a year in Scandinavia, studying the care and treatment of psychopathic offenders. This paper is based on observations and enquiries by the writer during that year.

It is not proposed to deal with out-patient treatment or use of probation, except to note that probation is being extended with benefit to habitual petty offenders in place of short-term sentences.

In none of these three countries is there legislation referring explicitly to "psychopathic" offenders as such. The provisions are for offenders suffering from mental abnormality short of certifiable insanity, thus covering a wide variety of conditions, but for convenience only the term "psychopathic offender" will be used here.

The provisions for psychopathic offenders in Norway, Sweden and Denmark differ in various important respects. and these differences are more comprehensible if viewed against an outline of the general historical development of this field. Such a valuable comparative historical review is given in the International Penal and Penitentiary Foundation's publication "Modern Methods of Penal Treatment", to which I am indebted.

Sixty to seventy years ago the Penal Codes of various countries started to include special penal sanctions, which differed in principle from other sanctions, and which came to be termed "security measures". They consisted in the replacement of punishment meted out according to the severity of the crime by a measure which made allowance for the nature and personality of the criminal. The aim of these measures was initially confined to the protection of society, and so-called "incorrigible" criminals were imprisoned for lengthy periods for that purpose, rather than for their own benefit or reformation.

The development of security measures proceeded in two directions, apparently based on the notion that those offenders who required "security measures" fell into one or other of two categories. First were those who were thought to show no significant mental abnormality—that is significant in the sense of wholly accounting for, or at least greatly contributing to their criminal behaviour without amounting to psychotic illness. The basic principle in dealing with this first category remained that of "preventive detention" i.e., simple internment for the protection of society.

Into the second category came those who did show significant mental abnormality; internment for reasons of social security was still necessary, termed "security detention", but the need for some kind of treatment or re-education was also recognized.

Up to the 1940's various countries, for example Sweden, approached the problem of re-education by stressing the importance of a finely detailed classification of offenders, and the importance of having institutions corresponding to the numerous sub-groups which such a classification produces. This tendency influenced general penal policy in that country and resulted in the development of numerous small institutions, each dealing with a relatively homogeneous group.

In more recent years, there have been other developments, which have reflected the influence of a more dynamic, psychologically orientated approach. Re-education had tended to mean a training in steady work habits, the teaching of some form of skilled work, e.g., tailoring, joinery, metal work, the provision of school and library facilities and so on. This kind of regime undoubtedly made detention more humane and constructive—that is to say less liable to induce apathy and to undermine what little initiative and capacity to take responsibility for himself the individual might have. But the understanding of persistent asocial and anti-social behaviour in terms of recognizable personality defects, of a disordered capacity for forming relationships, and of the development of abnormal attitudes has become more widely accepted, and the conception of "re-education" has become correspondingly broader and more realistic. Re-education has come to mean regarding the offender as a maladjusted personality who needs the opportunity, and the motivation, to change in his relationships with others and in his attitude towards himself. This notion of re-education has led to a swing away from the principle of small institutions dealing with relatively homogeneous groups. In such an institution the tendency is to have a regime which, although differing from that in other institutions, is applied more or less uniformly to all the inmates. Re-education, in the sense in which it now tends to be conceived, requires more of an individual approach, and institutions which are larger have certain advantages. A large heterogeneous group can be divided into relatively homogeneous subgroups, permitting a more flexible manipulation of the individual's environment within the one institution. This policy demands, however, a higher level of training of staff, and also more specialist categories such as psychiatrists, psychologists, social workers and teachers.

A corollary of the current conception of re-education is that what is achieved inside the institution is in a sense only a prelude to what the individual must achieve after leaving the institution. Increasing stress is therefore being laid on the importance of after-care work.

The provisions for psychopathic offenders in Norway, Sweden and Denmark will be considered from several different angles, viz., the relevant legislation, facilities for psychiatric examination and selection of cases, institutions designed specifically for these offenders, and after-care arrangements. There are also the provisions for those offenders serving a prison sentence who develop an abnormal mental state which needs more specialized handling than is possible under ordinary prison conditions. A final aspect is the detention of those habitual offenders who are not considered to show significant mental abnormality. Institutions in this last category, and particularly those dealing with habitual petty offenders, receive in practice some of the most pronounced cases of psychopathic inadequacy at a late stage.

Norway

In this large but very thinly populated country, most of the population live in small communities and lead an essentially rural existence. Norway, therefore, lacks the concentration of population which tends to be associated with a high crime rate. However, this may soon change, since the drift from the countryside into the towns, especially into Oslo, has accelerated rapidly in the last few years.

All psychopathic offenders who are likely to require special provisions are psychiatrically examined. In Oslo, the City prison has a thirty-six bed hospital unit, which is the only one of its kind in Norway. It is under the direction of a psychiatrist, deals with both physical and mental illness, and many of the psychiatric examinations of psychopathic offenders are also carried out there. In most other cases, the offender is examined in the prison where he is in custody, or, if the case is a minor one, in an out-patient department or as an in-patient in a civilian hospital. The Courts refer all psychiatric reports to the Forensic Medical Council, which is a statutory advisory body to which the Courts, government departments and local authorities can apply for advice on any forensic medical problem. The Forensic Medical Council does not see the offender, but as a rule simply satisfies itself that there are no serious defects in the presentation of the findings and that the recommended disposal is reasonably appropriate.

The provisions we are interested in here have had a chequered history in Norway. A relatively indeterminate sentence for particularly dangerous offenders was introduced as far back as 1902, but in the following years this measure was seldom used by the Courts. It was repealed in 1929, and replaced by legislation which is still in force. The Norwegian law provides for the preventive detention of offenders who have committed two or more serious offences and who are thought likely to commit further serious offences. Preventive detention does not replace punishment for the offence but follows on after the prison sentence has been served. While in preventive detention the offender can be released at any time by the Department of Justice. For five years after release he is under obligatory supervision which is conducted by one or other of the numerous private after-care organizations.

Preventive detention was extensively used in the first four years following its inception, but thereafter the numbers fell and a sentence to preventive detention has not been passed since 1954. The decline of this sanction was matched by an increasing use of "security detention", which was also introduced in 1929. Security detention is applicable to any offender whose mental state is such that further offences seem likely. The maximum period for which an offender can be detained is laid down by the Court. He can be discharged at any time within that period by the Norwegian Home Office, and is then under obligatory supervision until the end of his sentence. This, of course, has the drawback that the greater proportion of his time the offender spends in detention, and therefore, presumably, the greater his need for help in readjusting afterwards, the less after-care he receives. The most questionable feature of the Norwegian system is that the offender may also be sentenced to a term of imprisonment which he must serve before his detention and treatment as a mentally abnormal offender can begin. This practice is apparently based on the argument that for punishment to retain any value as a general deterrent, it should not be wholly escaped by those who are even partially responsible.

Since 1951 a special institution has been established for the detention and treatment of mentally abnormal offenders at Ila, a short distanct from Oslo. This is a modern building (1940), with accommodation for 110, including a twelve-bedded hospital unit. The ordinary accommodation consists of six sections of fifteen cells each, arranged so that the men in each section live as a freely mixing group, with their own day-room and hobby-room, where they can occupy themselves if they wish. It should be added that the cells are much larger and better furnished than the usual prison cell. The institution has well-equipped workshops, and there is also a school and a library. Near by the main institution is an open unit for sixteen men, where conditions are much freer. In addition to mentally abnormal offenders sentenced to detention there, Ila receives from the prisons some of those whose behaviour in prison is too disturbing and uncontrolled for them to be kept there; the rest of their sentence is served at Ila.

The principles of the regime at Ila are sound, so far as they go. Each offender is interviewed by the heads of the various departments in the institution, who assess his handicaps, assets and needs. The policy to be adopted towards his employment and general management in the institution is then provisionally formulated, discussed with him, and periodically reviewed thereafter.

In certain respects, however, this institution labours under severe handicaps. For instance, of those sent to Ila since it was opened, a third were mentally defective and another third were dull and backward. While a certain number of those who properly belong to an institution like Ila will inevitably be of limited intelligence, this extreme degree of overloading with defectives is a considerable drawback to devising a constructive regime. On the other hand, Ila is too small to take all those sentenced to security detention. In consequence, some remain in prison and a few go to a penal work camp, intended for habitual beggars and vagrants. This is a bad feature since neither place has a regime suited to these offenders' needs. Furthermore, there are two groups within its own clientele which the institution cannot deal with. The hospital unit is well equipped for cases of physical illness, but is unsuitable for the management of psychotic reactions. Such cases are occasionally transferred to the isolation cells, but for the most part are sent to the prison hospital in Oslo City Prison. Ila has only a wire fence around it, being originally built as a women's prison, and any dangerous offenders who attempt to escape—a group which one would think requires particularly skilled handling—are transferred to a maximum security prison. The inability to cope with these two groups is unfortunate. It is important, in an institution of this kind, that there should be continuity of relationship between inmate and staff throughout all phases of the inmate's mental disorder, and there should be resources for dealing with all possible crises. The problems and difficulties are fully recognized and plans for a new building at Ila, which should enable it to take the more dangerous offenders, are under consideration.

The administrative head of Ila is a prison-trained governor, who is responsible for the entire management, except in strictly medical issues. The uniformed staff have had an orthodox prison training. They have had little psychiatric or psychological teaching to help them to understand and take part in an approach based on understanding of the individual offender and his needs. Two psychiatrists are engaged full-time at Ila, but their influence is inevitably limited by the kind of difficulty which arises when there is dual control by the psychiatrist on the one hand, and the prison administrator on the other. Psychiatric work at Ila includes group-therapy, individual psychotherapy in a small number of selected cases, and the supervision of those sexual offenders who are castrated in the institution.

Ila has developed its own after-care organization, on the principle that the offender is more likely to accept guidance by someone who has known him throughout his detention, than by someone he meets for the first time when he is released. Only a small number of released offenders is now dealt with by outside after-care organizations. The majority are supervised by Ila's three social workers, and it is probably in this field that the psychiatric influence has been greatest.

The Report (1956) of a Norwegian Royal Commission on penal reform expressed many of the objections to the present Norwegian system. For example, the combination of punishment and security detention is rightly condemned, although the Commission is unable to relinquish the underlying principle completely. Its suggestion that the Court might fix the minimum as well as the maximum duration of detention would shorten the time in which discharge could be made dependent on the offender's conduct.

The institution at Ila is to be enlarged to receive all those sentenced to security detention, and its psychiatric staff increased to four, but the present dual control of the institution goes unquestioned. The Report recommends improved training for all prison staff. Those working at Ila, however, require more specialized instruction, and the proposed changes in training would not be adequate.

A point not stated explicitly, but implied, is that the offender has a right to know the maximum time he can be detained. The result may be a wholly arbitrary termination of after-care just when it is most needed.

Sweden

Arrangements for dealing with psychopathic offenders in Sweden are more elaborate, but then Sweden is a wealthier country, is more urbanized, and has the stimulus of a serious crime problem.

In Sweden, as in Norway, the psychiatric examination of offenders in custody is conducted, as far as possible, in a special observation unit. Such units are provided on a lavish scale, there being seven of them scattered throughout the country. The most important is that directed by Professor Rylander, situated within Stockholm Central Prison. This unit has seventy beds (including twelve for women), a staff of seven psychiatrists and five social workers, and is an active training centre for clinical forensic psychiatry. Near to one of the closed institutions for security detention is an eighty-five-bed prison psychiatric hospital which includes examination prior to sentence among its functions. The remaining observation centres are smaller, and are located mainly in prison hospitals. The two large units in particular are also used for the psychiatric treatment of convicted prisoners who have broken down while serving sentence. These do not include the out-patient clinics specially designated for examination of offenders.

The Swedish legislation governing institutional provisions for psychopathic offenders differs considerably from the Norwegian. For instance, when the Swedish Penal Code was revised in 1946, the issue of "responsibility" was abrogated (Rylander, 1955). Before this revision, the issue of responsibility was governed in theory by criteria corresponding to the McNaughten Rules. In practice, and in the absence of any capital punishment to complicate the issue, psychosis was accepted as proof of irresponsibility and the offender was dealt with then, as he is now, by committal to an ordinary mental hospital or to a special institution for the criminal insane.

Prior to revision of the Swedish Penal Code in 1946, offenders who had committed several offences of a more or less serious character, and who seemed to require special provisions were sentenced to preventive detention. Apart from specifying how serious the present and previous offences must have been to justify preventive detention, the only other criterion laid down was that the offender should be unlikely to respond to punishment. The Court fixed the minimum period of detention between the limits five-fifteen years; after this minimum period had been served the sentence then became indeterminate.

In the revised law, the category of "security detention" was introduced, but the quality of the person's offences was not so narrowly specified, and besides being unlikely to respond to punishment, the offender's mental state must be abnormal. Before imposing sentence the Court consults an advisory body, the Internment Board, which has a medical and lay membership (the lay membership being in the majority). The Court fixes the minimum period of detention between the limits one—twelve years. The sentence becomes indeterminate after this minimum period has elapsed, except in the case of a minimum of one year, when the offender must be released after four years.

The Internment Board alone decides when an offender is to be released.

The old category of preventive detention was retained in law but has not been used since 1946, so that for practical purposes one can say it has been replaced by security detention.

As mentioned earlier in this account, the policy favouring numerous small institutions has been pursued in Sweden, and this has applied also to institutions for security detention. There are, in fact, six such institutions widely scattered. Of these six, four are closed institutions, with from one hundred to one hundred and twenty detainees in each, and two are open, with twenty to thirty in each. The four closed institutions are graded according to strictness of security precautions and flexibility of regime, and detainees are allocated accordingly. All those in the open institutions have first spent some time in a closed one.

The difficulties in providing specialist staff with a training in psychiatry or psychology for such a scattered organization are great. The only co-ordinating factor is that all these institutions are under the direction of one man, who does succeed in imbuing his senior staff with a progressive outlook. The writer visited three institutions—two closed and one open—all of which have excellently equipped workshops, where it is possible for a man of exceptional industry to earn up to £3 a week. Of this, two-thirds are retained by the institution and paid out to the man on his discharge. Some of those in the open institutions and in the "top grade" closed ones have been granted a privilege accorded to ordinary prisoners—that of travelling home unaccompanied for a week-end every two months, of course at their own expense.

In the last few years the top-grade closed institution has been extended to include a so-called half-open unit—that is to say there is a guard there at night—and a wholly open unit. A start has thus been made in this particular institution towards a system of graded responsibility and privilege.

So far as psychotherapy is concerned, this is limited to a group-therapy session every ten days or so, conducted by one psychologist who has to cover all institutions. The absence of any psychiatric contribution is the more remarkable in view of the well-developed facilities for psychiatric examination of offenders, and means that psychotic and other severe disturbances cannot be dealt with in the institution concerned, but must be transferred.

Discharge from security detention is followed by a period on licence, for at least three years, and the Internment Board appoints whoever is to conduct after-care supervision. Such a large and thinly populated country presents particular difficulties as regards the organization of after-care. The closed institutions each have a social worker who deals with those in detention, and arranges accommodation and employment when they are released. Supervision of those on licence, however, is usually undertaken either by the Prison After-Care Association or by Probation Officers, who may visit the person before his release. On the whole, therefore, there is limited continuity between social work in the institutions and the subsequent after-care, but it is hoped to remedy this defect by providing more social workers in the institutions.

One of the most difficult questions in dealing with psychopathic offenders seems to be that of the individual having sufficient motivation for the development of new patterns of behaviour. In other words, he must have some motivation for active and genuine participation in whatever therapeutic regime has been created. The problem is how to foster this motivation and one of the main arguments in favour of the indeterminate sentence is that it can contribute something to this problem—if it is utilized in the right way. In this respect, the Swedish law has grave defects.

The Court can fix the minimum period of detention at anything up to twelve years and the Court's decision seems to be determined in practice by the gravity of the offence. It is, therefore, fundamentally an act of punishment, even though the law states that the offender must be unlikely to benefit from punishment. This contradiction in Swedish practice is a more serious defect than the Norwegian practice of combining punishment with security detention, since in Norway the Department of Justice can, if it chooses, waive the punishment.

The absence of any psychiatric contribution in security detention institutions has probably contributed to a movement in Sweden away from the indeterminate sentence and towards a more formal, legalistic conception of justice. The feeling behind this movement seems to be partly that, in the absence of a more positive and active re-educational or therapeutic regime, the indeterminate sentence is by its nature excessively harsh and ethically indefensible.

The Swedish law is likely to be changed in the near future, so that although the Court will still fix the minimum period of detention, an individual will not be detained for more than three years over the minimum, without the Court's sanction. The Court will then have the power to prolong the detention at three yearly intervals. This change will also limit the powers of the Internment Board, which at present has full control over discharges, and against whose decision the individual has no right of appeal. The criteria of mental abnormality and unresponsiveness to punishment will be discontinued. The offence leading to the new form of detention will have to be a serious one, ordinarily punishable by at least two years' imprisonment, a much heavier sentence by Swedish standards than it is in this country.

The course of events in Sweden exemplifies the uncertain groping which characterizes much of the development in this field. The lack of any definition of the nature of the problems and of the target to be aimed at is evident, but is not, of course, peculiar to Sweden. Confusion of policy, combined with a willingness to spend large sums in this field, seems to lead to excessive emphasis on the buildings and surroundings of an institution, and to insufficient stress on the provision of adequately trained staff. However, as Cornil points out in his preface to "Modern Methods of Penal Treatment", it is easier to talk of re-educating offenders than it is to formulate in detail just what the educators are supposed to do, how they are to do it, and how they themselves can best be trained.

Denmark

Facilities in Denmark for the psychiatric examination of offenders are of various kinds. In Copenhagen itself there are two observation units, each consisting of a small block of cells in the remand prison next to the City Court and each with a staff of psychiatrists and social workers. 2–3 per cent. of those examined in these units are further examined in a mental hospital or in one of the general hospital psychiatric units in Copenhagen. Elsewhere in Denmark the offender is reported on by the local M.O.H.—and some of their reports are very good. Some of these cases also may be further examined in a mental hospital. This practice of admitting offenders to mental hospitals for examination has been increasingly criticized in Denmark itself. Apart from physical investigations, it is doubtful whether the clinical picture ever becomes clearer, and the reverse may happen. These patients are also a source of difficulty to the mental hospital, and to get round this the mental hospital examination of the most disturbed offenders has recently been centralized in one hospital.

As in Norway there is an advisory Forensic Medical Council, to which the Courts can submit any medical report.

The development of security measures in Denmark dates from 1925. Up to that time, repeated offences were dealt with by increasingly long prison sentences. In the case of mentally abnormal offenders, the prison sentence was modified in accordance with what was considered to be the degree of responsibility. Dissatisfaction with these measures led to the appointment of a Commission which was to draft a new Penal Code, but an interim law was passed in 1925, before the Commission had completed its work. This law, which resulted from one particular case, permitted the detention, for an indeterminate time, of mentally abnormal persons who had committed sexual offences, incendiarism, and acts of violence, including murder.

The revised Penal Code became law in 1933 and established various special sanctions which have not been altered since then. Supposedly normal but dangerous habitual offenders can be sentenced to "preventive detention". The minimum period of detention is four years, each case is then reviewed by a special advisory board at two-yearly intervals, but detention for more than twenty years requires Court sanction.

Habitual petty offenders may be sentenced to detention in a penal work camp. This sentence is relatively indeterminate in that the offender's suitability for release must be kept under review after two years and he must in any event be released after five years.

In defining the sanctions applicable to psychopathic offenders, the Danish law does not attempt to define psychopathy. It refers to the offender displaying an abnormal mental state, not amounting to insanity, and due to "mental under-development, impairment or disturbance of the mental faculties, or sexual abnormality". The Court is to decide on the basis of a medical report and all other relevant circumstances, whether the accused is likely to respond to punishment.

If he is thought likely to respond to punishment he may serve a prison sentence either in an ordinary prison or in a prison for psychopaths. If considered unresponsive to punishment, he may be detained in an institution for psychopaths. But the prisons for psychopaths and the institutions for detention of psychopaths are in fact the same, the only difference being in the nature of the sentence. Detention is an entirely indeterminate sentence; the offender is released by the Court which originally sentenced him, and is then under obligatory supervision, also for an indeterminate period.

The extent to which these various forms of detention and imprisonment have been used has altered in the course of time. Sentences to imprisonment in an institution for psychopathic offenders were fairly common in the pre-war years but are now very rare. Since 1950, there has been a steady decline in sentences to preventive detention, which have now fallen to only one or two a year, and sentences to indeterminate detention in a special institution for psychopathic offenders have increased during the same period. The first of these institutions, the well-known one at Herstedvester, was opened in 1935 and in 1952 Horsens State Prison was converted into a second. Preventive detainees were segregated in one wing of Horsens Prison, but since the prison changed its status these offenders have been allowed to mix with the other inmates and are subject to the same regime.

Committal to the penal work camp is still used quite often, but this also has diminished since about 1950, an increasing proportion of those who would previously have gone there being sent instead to an institution for psychopathic offenders.

The institution for psychopaths at Herstedvester began with fifty offenders, many of whom had been sentenced under the interim law of 1925, and had therefore committed particularly serious offences. The statutory regulations governing Herstedvester state that its purpose is first the detention of those who are a serious danger to public law and order and second their treatment, but only insofar as treatment does not conflict with the protection of society. Herstedvester was built near a State Prison, a few miles to the west of Copenhagen. The Prison Governor was to be in administrative charge while a psychiatrist was in charge of "treatment", but their respective spheres were defined with a vagueness which left them ample opportunity to work out their respective roles between themselves. Initially, however, there was only a part-time psychiatrist, and it was not until 1942 that a full-time Medical Director, Dr. G. K. Stürup, was appointed. These details are mentioned because they show that the institution has had a "control period" when treatment had not yet been established. Also in those early years, the uniformed staff had all had only an orthodox prison training and only experienced guards were sent to Herstedvester. Inevitably, many of them found it difficult to adjust to an institution where the emphasis was supposed to be on treatment rather than punishment, even though the offenders they were dealing with were desperate characters. Most of the original guards have now gone, but those that are left can recall vividly their own, and also the detainees' confusion and resentment, and the frequent outbursts of violence.

In its appearance, Herstedvester is unmistakably a penal security institution. The detainees live in cells and practically all the windows have stout iron bars. In contrast to these external appearances, the atmosphere, which is that of an active and well-directed therapeutic institution, is all the more impressive.

The institution consists of several separate buildings, the main feature of which is that the cells are arranged in groups of 10 or 15—each group having its own day-room and a small work-room. There are twelve such cell-groups, and also a building for eighteen where the patients share rooms and where they run their own communal life with relatively little supervision. In addition, there is an open unit for thirty patients a few miles from the Institution, created out of two adjacent farms. Thus the principle of increasing flexibility of control over the patient's environment, by diversifying small groups within the one institution, is well illustrated at Herstedvester. On the same principle, the work facilities for the patients have been multiplied as far as possible, but, in spite of much improvisation, there is a serious shortage of suitable work.

In addition to this accommodation, there is a 14-bed hospital unit where psychotic reactions can be adequately managed. Immediately adjacent to the hospital unit are six isolation cells which have some notable features. They are the only ones seen by the writer which accord with the principle that isolation should be strictly a measure for the protection of the individual concerned and others, and not a vindictive retaliation. Instead of the usual gloomy dungeon, with minute panes of thick semi-opaque glass set far out of reach, the isolation cell at Herstedvester has larger windows than an ordinary cell, set lower down, and looking over one of the main thoroughfares of the institution.

In addition to the Medical Director, there are four psychiatrists, two psychologists, three nurses and *circa* 130 uniformed staff. For some years now the uniformed staff has been recruited by taking volunteers from new entrants to the Prison Service. These serve a probationary period of about a year at Herstedvester, followed by a three months' course at the National School for Prison Officers, and then those who are to return to Herstedvester spend three months as a mental hospital male nurse.

The various ways in which the therapeutic problems presented by psychopathic offenders are approached at Herstedvester can be mentioned only briefly here. Individual psychotherapy makes some contribution but, for various reasons, it must play a relatively subsidiary role except in a few cases. Group therapy figures rather more prominently, but even then, the majority of the 200 patients at Herstedvester, probably about 70 per cent.—are left uninvolved in any form of psychotherapy which is anything more than purely *ad hoc* support, and yet are at least as much in need of treatment as the rest. With the aim of trying to meet the overall need for treatment—i.e., the need of every patient—the main therapeutic effort goes into creating a therapeutic community. This work has developed independently of, for example, the work with therapeutic communities described by Maxwell Jones (1952), and differs in many respects from the latter, not least because of the different framework within which it has to be conducted. This approach—the attempt to create a therapeutic community with 200 psychopathic offenders with indeterminate sentences—is one of the most important of Herstedvester's contributions in this field. Another important contribution is the quality of the after-care work which is of a very high order. Discharge is always on licence, again for an indeterminate period, which in practice lasts three to five years. In case of relapse while on licence, a remarkable degree of co-operation between the institution and the Courts is often evident. Herstedvester has six social workers with a total case load of about 350 patients on licence.

If one applies the criterion of no recidivism for five years after the offender has returned to society, successful results have risen from 40 per cent. in the pre-war period, to over 60 per cent. in recent years. Such a simple comparison, however, is not valid without taking into account various factors such as a change in the type of offender treated, in that sexual and violent offenders now constitute a smaller proportion than they did in the pre-war period. The average duration of detention has diminished, which may be partly related to the change in type of offender, but an accurate quantitative assessment will depend on the statistical survey of Herstedvester now being prepared by Dr. Stürup and his colleagues.

A final point about Herstedvester is that prisoners who become too disturbed or too difficult to remain in prison may be transferred there, either returning to the prison after a short period, or serving the rest of their sentence in Herstedvester.

There is also a unit for women psychopathic offenders, contained in the one State Prison for women, some miles to the north of Copenhagen. The unit for psychopathic offenders has only about fifteen women, most of whom have committed particularly serious offences, such as assault, murder, and arson. Medically, this group is looked after by the Herstedvester staff, and their after-care is understaken by Herstedvester's social workers.

Discussion

The legislation of all three countries provides for both preventive detention and security detention, and a notable feature is how in actual practice the former has been replaced by the latter. The Courts have thus come to a tacit acceptance of the idea that repeated offences are in themselves sufficient evidence of a serious maladjustment which requires treatment. On the other hand, the legislation for security detention in Norway and Sweden is inconsistent since, either explicitly as in Norway or implicitly as in Sweden, it attempts to combine retribution for the offence with treatment for the mental abnormality which led to the offence. The Danish law provides simply for the detention and treatment of the offender until he is able to conduct his life more normally, and is thus based on the same principle which is implicit in the civil certification of the mentally ill. Even in Denmark, however, the conception that the sanction imposed by society must bear some relation to the gravity of the offence can, in practice, influence the Court's decision as to when an offender is to be discharged. Fortunately, the attitude of the Danish Courts is, on the whole, an enlightened one.

All three countries have special institutions for the detention and treatment of psychopathic offenders, but the extent to which psychiatry makes its proper contribution varies greatly from one country to another. It is not sufficient, as in Sweden, to be able to refer particularly difficult cases to a psychiatrist, nor, as in Norway, to insert the psychiatrist into an institution which is directed by a non-medical, penal administration and where he is restricted to the treatment of only some of the inmates. All the inmates suffer from a disorder which is the psychiatrist's province, and their need is more adequately met by the Danish system where the environment and day-to-day management of all inmates are under psychiatric direction. It is sometimes argued that for a psychiatrist to take complete charge of a penal institution involves him in an obligation to society, which he would tend to neglect when it conflicted with his medical obligations to his patients. He might, for example, recommend discharge when he considered it would be beneficial for the offender, even though society was thereby exposed to a considerable risk. In point of fact there need be no such conflict of loyalties. The interests of society and those of the offender are both best served by the offender being helped to make a more normal adjustment. The premature release of a psychopathic offender may be a serious matter for society, but it is more serious for the offender himself, since it exposes him to an unnecessary risk of relapse and to the deepening of feelings of fatalistic despair and isolation which relapse often provokes. While the interests of society do not in fact clash with the offender's, it is possible for the convenience of society to be favoured at the offender's expense by needlessly prolonged detention, and this is presumably less likely to be countenanced by the psychiatrist than by a non-medical director. The two special institutions at Herstedvester and Horsens in Denmark both have medical directors, and the writer could find no evidence of this arrangement working to the detriment of either the offender or society.

SUMMARY

The legislation concerning psychopathic offenders in Norway, Sweden and Denmark is reviewed in the light of the general trends in penal policy in recent decades. The facilities for the psychiatric examination of offenders in these three countries are described. The development of special institutions for the care and treatment of psychopathic offenders is described and discussed, with particular reference to the importance of the psychiatric contribution in this field.

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A PAIRED-ASSOCIATE LEARNING TEST FOR USE WITH ELDERLY PSYCHIATRIC PATIENTS

By

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In his examination of elderly patients the psychiatrist is commonly concerned to obtain some indication of the individual's "memory function". When the psychologist is asked to help in such assessment there are at least two factors which may handicap his ability to do so. Firstly, the conception of "memory". as popularly used, even in the clinic, is rather narrow in its connotation, having come to refer almost exclusively to the *reproduction* of learned material. It would seem more satisfactory, from the psychologist's point of view, to substitute instead the notion of "learning ability" since this expression has a wider meaning. It admits of the discussion of separate aspects and takes into account, for example, the two broad phases of "acquisition" and "retention". Secondly, those tests which the psychologist may have by him which purport to assess "memory" usually suffer from a number of disadvantages. For example, as Williams (12) has recently pointed out, they are seldom so constructed as to ensure that every subject has a standard opportunity to acquire what the test requires him to reproduce; in any case few of the tests available have ever been given to elderly people, so that "normal" performance in such age groups is unknown. A more important, and less often emphasized, disadvantage, however, is that performance on such tests is commonly closely related to performance on intelligence tests, so that although the ostensible content of the material used seems to be related to "memory", in the popular sense, what the psychologist in fact gets from them is another measure of general intellectual ability. A study by Hall (1) has shown that, for a young population at least (mean age 29.11, sd 2.77), the results of the Wechsler Memory Scale (10) correlate with results on the Wechsler Bellevue Intelligence Scale (9) almost to the degree ($r=0.77$) to which the intelligence measure correlates with itself ($r=0.94$).

Following a series of experiments designed to investigate "memory function" in elderly psychiatric patients the present author and his colleagues (Shapiro, Post, Lofving, and Inglis (8), Inglis (2)), have attempted to construct a test which would be (a) sensitive to memory impairment, and (b) as independent as possible of intellectual functioning. This paper will describe a test which has been evolved with these ends in view and will present some of the data which have been collected. It should be pointed out that, so far, the test cannot be regarded as a standardized test of memory in the sense that it could be used to order individuals along a continuum of memory ability in the same way that an intelligence test may be used to order individuals in terms of intellectual status. It can be shown, however, that it is a test of the "acquisition" phase of learning which is quite sensitive to *impairment of memory* in the elderly; and even this simple use may be of some service in the geriatric clinic, for example, in the objective description of a patient's symptoms, in the assessment of the effects of treatment, and possibly even in prognosis.

Test Material· The test itself is of the ordinary paired-associate learning type, employing verbal presentation and the simple recall form of reproduction. Two alternative sets of stimulus-response material (whose statistical equivalence has been demonstrated (Inglis (3)) have been used, as follows:

	Form A		*Form B*	
	Stimulus	*Response*	*Stimulus*	*Response*
(a)	Cabbage	Pen	Flower	Spark
(b)	Knife	Chimney	Table	River
(c)	Sponge	Trumpet	Bottle	Comb

The patient is given instructions much like those for the Paired Associate item of the Wechsler Memory Scale (10). He is told, "I am going to read you a list of words two at a time. Listen carefully because after I finish I shall expect you to remember the words that go together. For example, if the words were 'East—West', 'Gold—Silver', then when I said the word 'East' I should expect you to answer 'West', and when I said the word 'Gold' you would, of course, answer (pause) . . . 'Silver'. Do you understand? Now listen carefully to the list as I read it."

The examiner allows an interval of about 5 seconds between the pairs of words when reading the list. After the presentation of the list another 5-second interval is allowed. The stimulus words are then presented one by one in random order. Thus, the examiner asks, "What went with 'Flower'?" The patient is then allowed about 10 seconds to reply and if his answer is correct the examiner says "That's right". If the reply is wrong he says "No", and supplies the correct association. If no reply is given by the patient within about 10 seconds the correct response is again supplied by the examiner.

The material is presented in this way until the patient gets 3 consecutive correct responses for each stimulus word or until each stimulus word has been presented 30 times, whichever is sooner. The examiner stops presenting each stimulus as its criterion is reached. Supposing that one pair is learned to the criterion before the other two then the appropriate stimulus word is dropped out and the remaining pair are simply alternated.

The score on this test is the sum of the number of times the stimulus words are presented before the criterion is reached; for example, the score of the following patient would be 11.

	Initial presentation	*1*	*2*	*3*	*4*	*5*	*6*	*. 30*
a.	–	–	+	–	+	+	+	
b.	–	+	–	+	+	+		
c.	–	–	+	–	+	+	+	

It can be seen that, with this method of scoring, the minimum (i.e. the "best" score) is 3, and the maximum (or the "worst") score is 93.

Patients: The test has been given to 36 patients. These comprised an experimental group of 18 elderly persons clinically estimated to be suffering from undoubted memory disorder. This estimate was made by the consultant psychiatrist in charge of the Maudsley and Bethlem Royal Hospitals Geriatric Unit, Dr. Felix Post, on the basis of a full psychiatric investigation of each patient. The main considerations entering into his judgment were, (i) that evidence emerged during the doctor's examination that the patient's memory was impaired, and (ii) that the patient's relatives complained about his memory.

A control group of another 18 patients without memory disorder was also tested. Each of these was matched as closely as possible with a member of the experimental group in terms of chronological age and also in terms of the Verbal Scale Weighted Score (VSWS) as obtained from either the Wechsler-Bellevue Scale (9) or the Wechsler Adult Intelligence Scale (11). There were 7 men and 11 women in each group. The age range of the experimental group was 56 to 82, the age range of the control group was 58 to 81.

Results· The differences between the groups on the relevant variables (i.e. Age, VSWS, and the Auditory Recall (AR) score) were estimated by Sandler's A-test (7); the results secured are shown in Table 1.

TABLE I

Means, standard deviations, and the significance of the differences between the means of the criterion groups on Age, VSWS, and AR scores.

	Experimental Group		*Control Group*		*A*	*p*
	m	*sd*	*m*	*sd*		
Age	68·56	7·16	69·61	6·41	·507	NS
VSWS	33·50	8·73	32·83	7·10	4·500	NS
AR	59·00	25·06	13·00	6·16	·070	<·001

Since the distribution of scores on the learning test was not normal in both groups a histogrammatic representation of the scores obtained by members of these groups provides a more convenient description of the facts for clinical purposes (see Fig. 1).

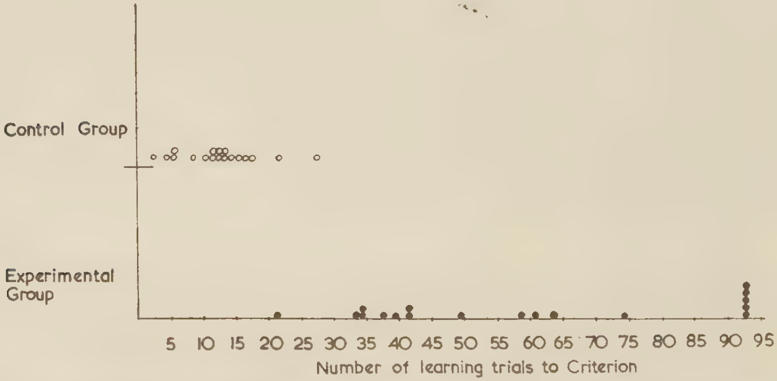

FIG. 1.—Raw data on the paired-associate learning test (AR) for both experimental and control groups.

It has already been pointed out that one disadvantage of many of the so-called "memory tests" currently available is that performance on them is closely related to performance on intelligence tests. To examine this aspect of the present test, rank-difference correlations (McNemar (6)) were run between the VSWS measure and the AR results, and also Age scores and AR results for both groups, the results being as follows:

Experimental Group:	VSWS × AR rho $= -0{\cdot}34$	Age × AR rho $= 0{\cdot}08$
Control Group:	VSWS × AR rho $= -0{\cdot}03$	Age × AR rho $= 0{\cdot}09$

None of these correlations approach an acceptable level of significance and we may therefore conclude that the AR test is testing something other than general intellectual ability, at least as far as this is measured by the Wechsler Verbal Scale.

The Auditory Recall test is currently being further assessed in:

(i) An experiment designed to secure a cross-validation of the effects of the vitamin preparation "Parentrovite" on memory function in elderly patients (Krawiecki, Couper and Walton (4)).

(ii) In a prognostic study of elderly psychiatric patients with "doubtful" memory disorder. In the latter study cases whose memory status is not clearcut are being tested and will be followed up psychiatrically, after an appropriate interval, in order to find out if the test has predictive as well as descriptive value.

(iii) It is also being used in an investigation to examine the claim that "unilateral ECT" (Lancaster, Steinert and Frost (5)) may produce less mental impairment than bilaterally induced seizures.

SUMMARY

A paired-associate learning test for use with elderly psychiatric patients has been outlined. The test has been shown to be of some value descriptively, and other aspects of its possible clinical usefulness are currently being tested.

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PARANOID SCHIZOPHRENIA IN A MONGOLOID DEFECTIVE: SOME THEORETICAL CONSIDERATIONS DERIVED FROM AN UNUSUAL CASE

By

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In the past thirty years a great deal of attention has been paid to the problems of differentiating mental deficiency from psychosis, and recognizing the distorting effects of mental defect and deprivation upon supervening mental illnesses. The incidence of psychotic syndromes in mental deficiency hospitals has been variously estimated as 5 per cent (Greene 1933), 8 per cent (Vanuxem 1935), and 11 per cent (James 1939), while a survey made by Hackbush (1935) on a very large number of patients on the waiting list for admission to mental deficiency hospitals, suggested a figure of 10 per cent. James considered that three-quarters of his psychotic group were schizophrenic, and Bergman *et al.* (1951) agreed that schizophrenic illnesses were very common in mental defectives. Whereas the incidence of schizophrenia in the general population is 0·85 per cent (Mayer-Gross *et al.* 1954), it occurs as much as nine times more frequently in certain mentally defective groups.

This differential incidence does not imply that a mentally retarded patient has, *ipso facto,* an increased vulnerability to schizophrenia. Mental deficiency hospitals tend to admit and retain those patients who are the most socially incapable, and the double handicap of defective intelligence plus a supervening psychosis clearly will call for long-term care. Moreover, the ranks of schizophrenic mentally defective patients may be swollen by those who developed schizophrenia in childhood and whose general behaviour simulates primary feeble-mindedness (Neuer 1947), particularly after the acute phase of the psychosis has subsided (or been forgotten) and been succeeded by a functional dementia. It may be objected that many apparently schizophrenic patterns occurring in defectives are basically severe neuroses, but it seems unsound to class as psychoneurotic such symptoms as grossly deteriorated behaviour, delusions and hallucinations. Even should the high rate of schizophrenic illness in mental deficiency hospitals not represent accurately the incidence in the total mentally retarded population, it seems remarkable that paranoid schizophrenia complicating mongolism has not up to now been described, although mongoloid defect is a condition that can occur as often as once in 700 births (Penrose 1949), and accounts for between 5 and 10 per cent of patients in British and American mental deficiency institutions (Wallin 1949).

Because of its apparent rarity, the interesting considerations raised by its pattern, as well as the encouraging response to physical treatment, the following case is reported.

CASE REPORT

Maurice, a 32 year old male mongoloid defective, was referred by his general practitioner in January 1957 because of severely disturbed behaviour over the previous five months. The patient's mother was an intelligent and reliable informant who gave a clear description of his illness.

History of present illness

Until August 1956 Maurice had been an affectionate, compliant mongol who lived with his mother in a comfortable home, obtaining a great deal of pleasure in playing with his toys, listening to the radio and watching television, and by visiting relatives and the cinema. He had loved his grandfather who had taken over the paternal rôle after the death of Maurice's father when the patient was a child. Following his grandfather's sudden death Maurice became anxious and depressed, and insisted on viewing the body. He remained depressed for two days; on the third there was an abrupt change in his behaviour. Formerly he had been timid, averse to strangers and the dark, mildly hypochrondriacal, and desirous of winning approval and affection. Now he became truculent, abusive and disobedient, and lost his shyness. He began systematically to destroy playthings that he had once cherished. Sleep was disturbed by initial insomnia and frequent waking; nocturnal masturbation that had occurred infrequently in the past now ceased. At times Maurice was discovered eating soap and paper.

He complained that "daft people" were insulting him and making mocking faces at the windows, that he was being whipped and pricked, and that his mother was plotting to put him in a "Home" and was giving away his possessions. Fears were expressed of wolves lurking in shadows and in cupboards, and of gangsters and "space-men" who were out to kill him. In response to fears that he was changing into a hairy animal or a "plastic man" Maurice began constantly examining his body for evidence of change, and washed his hands and face so often that eczema developed.

Despite the terrifying nature of his fears the patient's emotional state appeared generally calm and self-satisfied, but spells of weeping or laughter occurred at times without obvious cause.

Personal history

The patient was conceived when his mother was 22, and pregnancy was uneventful. Birth was by forceps delivery, the weight at birth was 7 lb. Although his general health was satisfactory, development was grossly retarded. He was deemed ineducable after attending school for a few weeks when five years. Until he entered hospital for treatment of his psychosis Maurice had never been parted from his mother for more than a few hours in the day. In view of Connell's findings (1958) that amphetamine intoxication may result in paranoid psychotic reactions, it should be remarked that this patient had never received amphetamine medication in any form.

Family history

This contained no relevant information.

Findings on examination

Maurice was a short pyknic mongol with a scaly dermatitis affecting hands and face. His rain-coat was worn inside out, and he brandished a toy pistol. He was suspicious and unco-operative, although his face was expressionless, and spoke in a monotonous American accent and with an assumed idiom. "Up with them mitts, Chief. Put 'em up. The game's up. I'm not getting a rubber face like you, Mister, I know you, Chief. Jim Darke, double-wise special space agent. Right? Right? Chief special scout, space-ways gang. Planet X. One peep out of you, Jim Darke, and you get this. Are you special agent 1234 . . .? Take off those glasses. They are my dad's glasses. They are my glasses. Let me see your face. What are you doing to my face? Take off that face. Now, off with it! What are you doing to my face? Are you a plastic man, Chief?" He accused the psychiatrist of turning him into an animal by means "of the needle", of mocking at him, and variously mis-identified the doctor as one character or another derived from crime and science fiction. His confusion extended to his own identity, and he seemed unable to distinguish reality from garbled memories of film and television entertainment. He knew his address and the day of the week, but described the clinic as "a police station, a space station". He covered pages of paper with stereotyped scribbles indistinguishable from the manneristic writing of schizophrenics with normal levels of intelligence and education.

Psychological tests

Attempts to use psychometric and projective tests were unsuccessful. The pre-psychotic social age, as assessed on the 1936 revision of the Vineland Social Maturity scale, was 6·4 years.

Diagnostic formulation

On the basis of evidence of visual, aural, and tactile hallucinatory experiences, delusional ideas of persecution and self-transformation resulting in obsessive-compulsive rituals, inability to distinguish reality from fantasy, perseveration and stereotypy of ideas, and the impoverishment and incongruity of affect, it was considered that a florid paranoid psychosis had developed in this mongolian defective patient.

It seemed probable that the psychosis had been precipitated by the stress of losing a father-substitute, and a reactive depressive state; these were likely to recall painful memories of his own father's sudden death and to awaken intolerable feelings of insecurity and threat. But the character of the ensuing psychosis did not suggest a basic depressive illness.

Treatment and progress

Maurice was admitted under the care of Professor Martin Roth to the Psychiatric Unit of the Newcastle General Hospital and given six electroplexies. A marked and sustained improvement followed in his mental condition, and he was discharged home after eleven days, taking 25 mgms. chlorpromazine hydrochloride t.d.s. This drug was continued for seventeen months, and then withdrawn without any recurrence of disordered behaviour. His present condition, over two years after the onset of an acute paranoid illness, suggests that remission is almost complete, and Maurice's behaviour at home is best described by extracts taken from a letter written by his mother.

"I have been in hospital myself, and I was surprised when I came home to see the affection he gave me. He often came and kissed me, and said "I like you." The only strange behaviour I have noticed is, if he thinks he is on his own, he will talk to himself and laugh just like one does at hearing a good joke . . . but all told he's very good and happy, and even sees a joke before me at times. He's on good terms with all the folks around . . . they always give him a word and have a joke with him."

DISCUSSION

(a) *Character of psychotic syndrome*

Apart from its rarity, this patient's illness was noteworthy for its well-marked classical symptomatology, although it occurred in a patient whose pre-psychotic mental level was that of an imbecile. It is tempting to suggest that the floridity and systemization of his thought disorder reflected credit upon the rich variety of experiences that he had previously encountered in everyday life. Had the environment been less stimulating it is likely that a supervening psychosis would have been impoverished for lack of cultural material, and then would have, *faute de mieux*, presented atypically.

(b) *Schizophrenic illness, severe mental defect, and mongolism*

Kallman *et al.* (1941) concluded from their extensive studies on twins that separate genetic complexes existed for endogenous types of psychosis and mental deficiency, and that the occurrence of both conditions in the one individual resulted from his inheritance of two genetic groups. If this is so, the incidence of paranoid schizophrenia in adult mongols should be much the same as that in the general population. The rarity of the case I have reported may be more apparent than real, unless it be postulated that mongolism and paranoid states are antagonistic conditions. Whatever the aetiology of mongolism, it is difficult to conceive that the condition should protect against the appearance of paranoid psychosis in adult life, apart from its high associated death rate in early childhood (Penrose). I suspect that paranoid schizophrenia can occur in disturbed mongoloid defectives, but is not recognized for lack of well-differentiated symptoms. Pearson (1938), Hayman (1939), and Herskovitz *et al.* (1941) have described the difficulties of diagnosing psychotic syndromes when a patient's I.Q. is below 50. Since institutional mongols tend to have a range of I.Q. between 19 and 63, with an average score of 36 (Wallin), the paranoid mongol would be expected to display bizarre mental symptoms instead of a recognizable thought disorder.

If catatonic psychosis be considered (admittedly crudely) as a schizophrenic condition primarily affecting volition and motor behaviour, and endogenous paranoid psychosis as one in which perceptual and thought disorder predominate, then it might be predicted that a schizophrenic mongol, because of severely defective intelligence and lack of environmental, social and cultural stimulation in his experience of institutional life, would not possess an adequate store of ideas and attitudes to furnish him with material for hallucinations, delusions, or a grandiose/persecutory frame of reference. His psychosis would tend to present as a catatonic state and/or a severe diffuse conduct disorder. The work of Rollin (1946) seems to support this contention since this author has succeeded in describing a "primitive" type of catatonic schizophrenia in a group of disturbed mongols.

Strickland (1954) suggested that mental functioning in mongols might be improved by a good and stimulating upbringing. Whatever the truth of this supposition, I think it likely that Maurice's paranoid illness owed its classical features to the excellent cultural opportunities that he had enjoyed in the past, despite his imbecile level. Without a substrate of ideas and a sufficient number of social experiences in the past, it is difficult to imagine how a thought disorder could flourish. Is it not possible that increased longevity and improved care of mongoloid defectives may result in further unequivocal cases of paranoid psychosis, and possibly of other forms of schizophrenia, being recognized in mentally-ill mongolian imbeciles and regarded as meriting as intensive treatment as is accorded the schizophrenic with adequate intelligence?

SUMMARY

A case of typical paranoid schizophrenia is described in a 32 year old mongoloid imbecile. Response to E.C.T. and a prolonged course of chlorpromazine was highly satisfactory.

It is suggested that the classical pattern of the psychosis reflected and resulted from the patient's excellent pre-psychotic social and cultural opportunities, and that typical forms of paranoid schizophrenia may be recognized more frequently in the future in mongoloid adults who have had adequate social stimulation.

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A CLINICAL TRIAL WITH A NEW ANTICONVULSANT DRUG, α-(P-AMINOPHENYL)-α-ETHYL GLUTARIMIDE PREPARATION 16038 (CIBA)

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Derivatives of glutarimide are known to exert an effect upon the central nervous system. To name only two which are widely used for clinical purposes: Glutethimide ("Doriden") is a sedative, Bemigride ("Megimide") a stimulant. Preparation 16038 (Ciba) α-(p-aminophenyl)-α-ethyl glutarimide, in animal experiments had shown properties which suggested its use as an anticonvulsant, and this paper is an account of a clinical trial with the substance on in- and out-patients suffering from epilepsy.

In this trial, three groups of patients were investigated:

1. Twenty-seven male patients resident in hospital, aged 28 to 71 years. Of these 27 patients, 20 completed 5 and 6 months respectively of the trial and 7 discontinued for various reasons to be discussed later. The average age of the 20 patients was 59·6 years, that of the group of 7 patients 50 years. The 27 men represented all the patients in the male wards of the hospital suffering from epilepsy.

2. Ten female patients resident in hospital. All these patients completed 4 and 6 months respectively of the trial with Prep. 16038. Their ages ranged from 28 to 70 years with an average of 57·7 years. These 10 patients were selected mainly as a group with as few seizures as possible but included one patient with 26 seizures in the 6 months preceding the trial.

3. Fourteen out-patients attending an epilepsy clinic at the hospital, 6 male and 8 female. These patients were selected on account of the difficulty of controlling their seizures while at the same time being sufficiently co-operative and/or under supervision to provide reliable data. Four male and 4 female patients discontinued the trial for various reasons which will be discussed later. The ages of the 14 out-patients ranged from 12 to 55 years with an average of 38 years. The ages of the 4 male and the 2 female patients who continued the trial for up to 9 months ranged from 12 to 50 years, average 27·5 years.

The total number of patients who took part in the trial with Prep. 16038 was 51 of whom 37 continued for a sufficiently long period to allow comparison between the results of routine treatment and of the new compound which was given either alone, or more often, together with other drugs.

METHOD OF ADMINISTRATION

Preparation 16038 was supplied in tablets of 250 mg. each. The change-over from the previous to the new medication was made gradually, one 250 mg. tablet of Prep. 16038 being given every two days in place of one unit used in the routine treatment until after 4–7 days the new treatment became established.

In 9 of the 27 male in-patients the drug replaced by Prep. 16038, 1,000–1,500 mg. daily was phenobarbitone in doses of 50–195 mg. daily. Thirteen of the patients had been receiving phenytoin sodium 100 mg., 2–3 times daily together with phenobarbitone sodium 50 mg. 2–3 times daily, and in 7 of these patients phenytoin sodium was continued in the previous dosage together with Prep. 16038. In 6 cases Prep. 16038, 1,000–2,000 mg. daily, replaced the phenytoin sodium and phenobarbitone sodium compound ("compound E+P") entirely. The remaining 5 patients formed a mixed group treated with phenyl-methyl-barbituric acid, with "compound E+P" and primidone, and one patient had not been receiving anticonvulsant treatment for several years. Of the 7 male patients in this group who did not complete the trial, 5 had been treated with "compound E+P", 3 capsules daily, one had been receiving phenobarbitone 98 mg. daily, and one phenyl-methyl-barbituric acid and "compound E+P". Preparation 16038 replaced phenobarbitone and/or "compound E+P" in the usual manner in the first 6 patients, the last one continued phenyl-methyl-barbituric acid and Prep. 16038 replaced "compound E+P".

The 10 female in-patients received a greater number of different drugs before being given Prep. 16038, usually including phenobarbitone, phenytoin, primidone, reserpine, chlorpromazine, and occasionally sodium amylobarbitone in different combinations. The medication of one patient only in this group is directly comparable with that of the male in-patients. This patient received "compound E+P" three times daily. Preparation 16038 in amounts of 500 to 750 mg. daily replaced one of the components of these complex schemes of medication, usually phenobarbitone or primidone.

Similar complex schemes of treatment but without tranquillizers were in use in many of the epilepsy clinic out-patients. Preparation 16038 in daily doses from 250 to 1,500 mg. replaced phenobarbitone, methoin, primidone, troxidone, phensuximide, and acetazolamide. Details are given in Tables IV and V.

NUMBER OF SEIZURES

1. Twenty male in-patients who completed 5 and 6 months of the trial.

In 19 of these patients the number of seizures in the 5 months immediately preceding the administration of Prep. 16038 was compared with the number of seizures while receiving the drug for 5 months. In one case the periods were 6 and 6 months. The total number of seizures in the months preceding Prep. 16038 was 114 while the total number of seizures during the administration of Prep. 16038 was 337 (see Table I). Equal numbers or fewer seizures while on Prep. 16038 were observed in 7 out of the 20 male patients. Three of them had had no seizures for more than 5 months and one had been without medication for many years and showed a reduction from 4 seizures to one. Among the remaining 13 who showed an increased number of seizures during the trial, two had been free from seizures for more than 6 months and all had been on anticonvulsant medication.

2. Ten female in-patients who completed 4 and 6 months of the trial.

The number of seizures of the 8 patients who completed 6 months was: before Prep. 16038, 11 seizures, during Prep. 16038, 30 seizures. The two patients who received the drug for 4 months only on account of an interruption due to the appearance of a skin eruption, had 35 seizures before and 18 seizures

TABLE I

Male In-Patients Who Completed the Trial

Name	Age	Seizures Before Prep. 16038	Seizures During Prep. 16038	Seizures After Prep. 16038
Cl. Ga.	28	25	22	6
Lo. Wa.	30	4	0	0
J.E.W.	33	6	37	9
Ew. Du.	36	1	11	—
Jo. Dav.	37	4	9	6
Ge. Has.	37	15	43	1
G. Cl. B.	37	1	59	0
A.J.W.*	45	29	25	23
Wi. Ra.	46	0	13	0
Fr. Ga.	47	8	33	22
Er. Bu.	50	0	17	0
C.A.B.*	57	0	0	0
J.S.H.	57	0	0	0
Ge. Ke.*	57	1	2	1
J.W.R.J.*	60	3	10	0
Wa. El.	64	1	19	2
Ge. Gr.*	64	4	1	3
Alf. S.	67	8	20	9
H.L. Sm.*	69	4	16	2
R.J.P.*	71	0	0	0
	Years	Total	Total	Total
Mean	49·6	114	337	84

* Patients marked by asterisk were given Prep. 16038 alone without addition of other anticonvulsants.

TABLE II

Male In-Patients. Trial not Completed

Name	Age	Seizures Before Prep. 16038 in 5 Months	Time and Cause for Interrupting Trial	Previous Medication Daily Amounts	Medication at Time of Interrupting Trial Daily Amounts
Jo. Du.	34	4	25 days. Status epilepticus	Phenytoin sod. 300 mg. Phenobarbitone sod. 150 mg.	Prep. 16038, 1,750 mg.
Er. Hac.	40	0	Paranoid patient. Refuses to take drug on 2nd day		
G.H. Sp.	42	25	27 days. Status epilepticus	Phenytoin sod. 350 mg. Phenobarbitone sod. 150 mg.	Prep. 16038, 1,000 mg. Phenytoin sod. 200 mg.
Ha. Co.	52	0	Died of pneumonia after 2½ months. No seizures		
Wi. Sh.	53	0	12 days. 1 seizure Patient unsteady, unable to walk. Chronic perinephric abscess		
Ha. Wha.	64		Pyrexia after 10 days. Died of pneumonia and chronic renal failure		
Al. Ba.	67		Died of carcinoma of the bladder 3 months after starting Prep. 16038		

Mean = 50 years.

during the administration of Prep. 16038. This makes a total of 46 seizures before and 48 seizures during treatment with the drug. Among these 10 patients, 5 had been free from seizures for more than 6 months. Three remained free and 2 had one seizure each while on Prep. 16038, one in the first week, the other one in the 11th week.

3. Six out-patients who completed the trial.

To obtain a degree of uniformity the number of seizures in this group is given for 6 months. No patient was observed for less than 6 months but 5 of the patients continued the medication for 9 months and in these cases two-thirds of the total number of seizures is entered (see Table IV). The number of seizures before treatment with Prep. 16038 was 323, during treatment it was 278. These figures are, however, not quite accurate because the boy of 12 in this group (St. V.W.) suffering from Sturge-Weber syndrome, apart from the attacks that could be counted by his mother, had very large numbers of short attacks lasting one to two seconds in which he would drop a cup or spill soup from his spoon or stumble against the walls of the room. According to his mother, these very short attacks increased from 10–12 per day to 30–40 or more.

4. Seven male in-patients who did not complete the trial (see Table II).

Three patients died of intercurrent or pre-existing illness: one of a carcinoma of the bladder, one of chronic renal disease, one of broncho-pneumonia. One patient with chronic perinephric abscess became very drowsy and unsteady within a few days after 2 seizures and had to be taken off the drug for this reason. One patient refused to take the drug on account of paranoid delusions. Two patients passed into status epilepticus after large numbers of convulsive seizures. Treatment with Prep. 16038 was discontinued after 24 and 25 days respectively. Both the patients recovered.

5. Eight out-patients who did not complete the trial (see Table V).

Of the 4 male patients, Tr. De., a boy of 12, fell ill with a febrile illness, probably influenza, and his mother stopped the drug when he had 2 seizures. Ha. Be. who had had 6 seizures in the 6 months preceding the trial had 8 seizures in 11 days when 65 mg. of phenobarbitone were replaced by 500 mg. of Prep. 16038. Er. Ha. having had nocturnal seizures only for several years suffered 3 seizures in daytime while at work and complained of feeling "dopey" when at first 500 mg. of Prep. 16038 were given in addition to his routine medication. Reducing routine medication to the amounts shown in the last column of Table V while continuing 500 mg. of Prep. 16038 relieved him of the "dopiness" but caused a further 5 seizures while at work. The trial had to be abandoned in this case because the patient was afraid of losing his job. Jo. Ta. passed into a clouded and agitated paranoid state within a day or two. This had happened periodically and in an unpredictable manner for many years under a great number of different drugs.

Of the 4 female patients, J.V. Pr. had had 12 seizures in the 6 months preceding the trial and suffered 26 seizures in 10 days out of the 14 during which she replaced 250 mg. of acetazolamide by 500 mg. of Prep. 16038. D.M. Jo. with 2 seizures in the 6 months before the trial suffered 3 and 17 seizures during the 2½ months of medication when two consecutive attempts were made to replace "compound E+P" by 500 mg. of Prep. 16038. Do. Woo. developed a morbilliform rash after 11 days on 500 mg. daily of Prep. 16038 replacing 600 mg. of troxidone daily, and could not be persuaded to continue medication with the new drug. Co. Gr. had had 20 seizures in all in the preceding 6 months and suffered 16 convulsive seizures as well as very numerous small absences in the last 6 weeks of the 2½ months under Prep. 16038 so that she had to go off work.

Relation Between EEG and Response to Prep. 16038

With the exception of one female in-patient who was too unco-operative, all patients in this trial had their EEG tracings investigated before starting

TABLE III

Female In-Patients

Name	Age	Seizures Before Prep. 16038	Seizures During Prep. 16038
Ma. Ha.	28	2	6
Ev. Ea.	34	0	1
Iv. To.	36	0	0
Ol. Wi.	43	24	18
Win. Bl.	51	1	13
F.H. El.	57	0	0
Hi. Col.	57	11	0
Ev. Ha.	62	0	1
El. Dou.	69	0	0
Fl. Ja.	70	8	9
	Years	Total	Total
Mean	57·7	46	48

TABLE IV

Epilepsy Clinic Out-Patients. Male and Female. Trial Completed

Name	Age	Seizures Before Prep. 16038 in 6 Months	Seizures During Prep. 16038 in 6 Months	Previous Medication Daily Amounts	Medication at End of Trial
St. V.W.	12	146	106++	Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Phenobarbitone 32 mg. Phensuximide 1,000 mg.	Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Phensuximide 1,000 mg. Prep. 16038, 1,000 mg.
De. Te.	27	60	40	Phenytoin sod. 300 mg. Phenobarbitone sod. 150 mg. Primidone 1,000 mg.	Phenytoin sod. 300 mg. Phenobarbitone sod. 150 mg. Primidone 750 mg. Prep. 16038, 1,000 mg.
Er. Ru.	28	55	41	Phenytoin sod. 300 mg. Phenobarbitone sod. 150 mg. Methoin 300 mg.	Phenytoin sod. 400 mg. Phenobarbitone sod. 200 mg. Prep. 16038, 1,500 mg.
Ar. Whe.	50	33	48	Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Methoin 200 mg. Primidone 500 mg.	Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Primidone 500 mg. Prep. 16038, 1,500 mg.
Pat. Cl.	20	3	>1	Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Phenobarbitone 32 mg.	Prep. 16038, 750 mg.
Jo Be.	28	26	42	Primidone 500 mg. Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg.	Primidone 250 mg. Phenytoin sod. 200 mg. Prep. 16038, 1,500 mg.
Mean	27·5	323	278		

the new drug, many of them repeatedly over several years, and in some cases under activation with intravenous sodium thiopentone and chlorpromazine. All patients who completed the trial were re-examined towards the end, usually in the last 2 weeks. No follow-up examination was possible in most of those who did not terminate the trial either because the general condition of the patient was too serious or because they had stopped taking the drug on their own without previous consultation and before attending the clinic for a report. But in all these cases, too, the basic EEG was well known. Improvement of the EEG in the following paragraphs is meant to indicate less frequent occurrence of paroxysmal features, restriction of the area where paroxysmal or other abnormal features are seen, or the replacement of abnormal by normal frequencies. Temporal lobe cases are patients showing spikes in the temporal areas in one or both hemispheres as the main abnormality.

Among the 20 male in-patients, 14 showed either no change or improvement of the EEG. Four of these were temporal lobe cases. In 6 patients the EEG deteriorated and 5 of these were temporal lobe cases. No other significant

TABLE V

Epilepsy Clinic Out-Patients. Male and Female. Trial not Completed

Name	Age	Seizures Before Prep. 16038 in 6 Months	Time and Cause for Interrupting Trial	Previous Medication Daily Amounts	Medication at Time of Interrupting Trial Daily Amounts
Tr. De.	12	2	3 weeks. Intercurrent infection ? Influenza 2 seizures	Phenytoin sod. 200 mg Phenobarbitone sod. 100 mg. Methoin 100 mg.	Phenytoin sod. 100 mg. Phenobarbitone sod. 50 mg. Methoin 100 mg. Prep. 16038 250 mg.
Ha. Be.	38	6	11 days. 8 seizures	Primidone 500 mg. Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Phenobarbitone 65 mg Acetazolamide 750 mg.	Primidone 500 mg. Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Prep. 16038 500 mg. Acetazolamide 750 mg.
Er. Ha.	48	45	1½ months. "Dopey." 8 seizures during working hours	Phenytoin sod. 300 mg. Phenobarbitone sod. 150 mg. Primidone 750 mg.	Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Primidone 500 mg. Prep. 16038 500 mg.
Jo. Ta.	55	Sudden development of a clouded paranoid state. Unfit for clinical trial.			
J.V. Pr.	25	12	2 weeks. 26 seizures in 10 days	Phenytoin sod. 300 mg Phenobarbitone sod. 150 mg. Acetazolamide 250 mg. D-amphetamine 5 mg.	Phenytoin sod. 300 mg. Prep. 16038 500 mg. Phenobarbitone sod. 150 mg. D-amphetamine 5 mg.
D.M. Jo.	28	2	2½ months. 20 seizures	Phenytoin sod. 200 mg. Phenobarbitone sod. 100 mg. Primidone 750 mg.	Prep. 16038 500 mg. Primidone 750 mg.
Do. Woo.	31	Numerous facial twitchings	11 days. Skin eruption	Phenobarbitone sod. 100 mg Phenytoin sod. 200 mg. Troxidone 600 mg	Phenobarbitone 100 mg. Phenytoin sod. 200 mg. Prep. 16038 500 mg.
Co. Gr.	37	20	2½ months. 16++ seizures. Inability to go to work	Phenytoin sod. 300 mg Phenobarbitone sod. 150 mg. Methoin 200 mg. Phensuximide 500 mg.	Phenytoin sod. 300 mg. Phenobarbitone sod. 150 mg. Methoin 200 mg. Prep. 16038 500 mg.

Mean=38 years.

TABLE VI

EEG		Number of Seizures Before Prep. 16038	Number of Seizures During Prep. 16038		Number of Seizures Before Prep. 16038	Number of Seizures During Prep. 16038
Unchanged or improved	Ar. Whe.	33	48	Pat. Cl.	3	1
	De. Te.	60	40	Jo. Be.	26	42
Deteriorated	St. V.W.	146	106++			
	Er. Ru.	55	41			

correlation between changes in the EEG and particular features in the recordings could be seen in these 20 cases.

In 6 of the 9 female in-patients whose EEG was investigated, the recordings were either unchanged or showed improvement. In 3 cases there was deterioration. There was one temporal lobe case in each group.

Among 4 male out-patients the EEG remained unchanged in 1, improved in 1 (a temporal lobe case), and deteriorated in 2. Of the 2 female out-patients who completed the trial one showed no change in the EEG, one showed improvement (see Table VI).

The basic EEG of the patients who did not complete the trial did not show particular features which would set them aside from the recordings of those patients who were able to complete it.

SIDE EFFECTS

Drowsiness was present to a marked extent in a number of male in-patients who became unsteady, took to their beds, became confused, irritable, and sometimes aggressive probably on account of the confused state. Drowsiness was noticed also among several of the epilepsy clinic out-patients.

Skin eruptions were observed in 2 male in-patients, 2 female in-patients and one female out-patient. In the 3 female patients the rash was discrete (morbilliform), appeared on the 9th to 12th day and subsided within 4–6 days. No treatment apart from stopping Prep. 16038 was applied in the case of the in-patients while the out-patient was given an anti-histaminic compound against the itching. Resumption of treatment with Prep. 16038 after 17 days and 2 months respectively did not cause a return of the eruption of the 2 in-patients. The out-patient refused to take the drug again. The 2 male in-patients showed erythema with large urticarial weals, one 14 days, the other 21 days after starting Prep. 16038. In both the cases the rash subsided within 6–8 days and medication was resumed 16 to 21 days after stopping it without ill effects.

Reduction of Benedict's solution by the urine of the patients who had been on doses of 1,000 mg. or more per day for about 2 months was observed in a high proportion of cases. The reduction did not appear to be due to glucose because a specific enzymatic test ("Clinistix") was consistently negative. The substance responsible for the reduction could not be identified. The possibility that it might be related to or identical with glycuronic acid was considered. Reduction of Benedict's solution disappeared after discontinuing Prep. 16038.

No other changes in the urine or in the blood picture which was investigated in all patients at various times, were seen. Chemical constituents of the blood were not investigated because there were no indications on clinical grounds.

BEHAVIOUR OF THE PATIENTS

Apart from occasional conduct disorder due to drowsiness as described, and a state of great emotional lability in one of the two patients with status epilepticus (which was accompanied by profound but temporary changes in the EEG), no consistent picture emerged from personal observations or from reports by other doctors, nurses, or relatives of the patients. In those patients who had sufficient insight to note changes of their mood, optimistic or pessimistic attitudes usually varied with the number and severity of seizures.

FOLLOW-UP OF 20 MALE IN-PATIENTS AFTER TERMINATION OF THE TRIAL

Table I shows details of the numbers of seizures in the 5 months immediately following the termination of medication with Prep. 16038 and return to previous medication. One patient of this group had a partial right-sided temporal lobectomy and has improved as far as violent and aggressive outbursts are concerned. The remaining 19 patients were put back on their previous medication in stages in the same way as that adopted 5 months earlier to introduce Prep. 16038. In one of the patients the number of seizures increased from 1 to 3, two remained free from seizures and 16 showed a reduction of the numbers of seizures. The total number of seizures of the 19 patients reviewed was 326 during the 5 months of the trial and 83 in the following 5 months.

DISCUSSION

Method of Changing from Routine Treatment to Prep. 16038 and Back

The method used here with one exception, i.e. the stepwise replacement over 4–7 days according to dose was adopted because it avoids increasing the number of units of drugs to be taken which arises if the drug under investigation is first added to the routine medication. This was of importance in many of the in-patients as well as out-patients: in the former case because some of the patients were rather reluctant to accept a change of the medication to which they were accustomed during the first one or two weeks, in the latter on account of the large number of units of drugs already taken.

Dosage of Prep. 16038

The largest daily amount of the drug in this series was 2,000 mg. Usually 1,000–1,500 mg. per day were given. This represented weight for weight usually 5–10 times the amount of the drugs it replaced, mainly phenobarbitone and "compound E+P". Higher doses were not used on account of the side effects observed, i.e. drowsiness and confusion, and skin changes. Amounts less than 1,000 mg. daily were given in the group of out-patients who did not complete the trial. This point will be discussed in the next paragraph.

Number of Seizures Before, During and After Prep. 16038

The most disappointing result is seen in the group of male in-patients, the number of seizures rising from 144 to 337 during medication to fall again to 83 in the 5 months after termination of the drug and return to routine medication. Of the 7 patients who did not complete the trial, two developed status epilepticus. The group of 10 female in-patients shows a more favourable result. It is, however, difficult to compare the two groups because in the male group, Prep. 16038 was as a rule at least half of the active medication whilst in the female group Prep. 16038 was usually the 4th or 5th active substance taken. It should also be noted that 5 of the 10 female in-patients had been free from seizures for many years with EEGs showing very few paroxysmal features. Among the epilepsy clinic out-patients those who completed the trial show a fairly favourable picture as far as numbers of seizures are concerned. However, in the case of St. V.W. (*aet.* 12) only the more prolonged attacks could be counted while short myoclonic jerks with or without clouding of consciousness were uncountable. One of the male and 3 of the female patients who had been in this group discontinued the drug on account of an increase of the number of seizures. One female patient (Pa. Cl.) improved very markedly and can be considered a full success. One male patient (Er. Ru.) felt that he had improved moderately. As mentioned above the out-patients who did not continue the clinical trial received less than 1,000 mg. of Prep. 16038 per day and it may well be that this lower dosage was responsible for the failure in some of them. It is, however, difficult to obtain a clear picture of the "sufficient" dosage from this trial because the two male in-patients who developed status epilepticus received 1,750 and 1,000 mg. per day respectively and the increased number of seizures of the male in-patients as a whole occurred with a dosage of 1,000 to 2,000 mg. daily. One gains the impression from this trial that 1,000 mg. or more per day is required to replace the more common anticonvulsant drugs used in conventional amounts.

EEG Changes During Treatment with Prep. 16038

Nothing of importance resulted from comparing recordings taken before and during treatment apart from the observation that among the male in-patients who showed deterioration of the EEG, the proportion of temporal lobe cases was rather high. The prognostic value of the EEG as far as an effect upon numbers of seizures is concerned seems to be about the same as that of a survey of the number of seizures during routine treatment: patients free from seizures or with few seizures for long periods had few paroxysmal signs in the EEG and did not increase the number of seizures under Prep. 16038 to the same extent as those with more seizures before using Prep. 16038 and a more irregular EEG. If no reliable data as to numbers of seizures are available, an EEG can therefore give valuable indications for the prospects of a success with the drug.

The Side Effects

Drowsiness observed during medication with Prep. 16038 was not of great importance among the in-patients who were under adequate nursing supervision. It was more disturbing for the out-patients and caused unfavourable comments. However, none of the patients refused to continue the drug for that reason alone. The skin eruptions were harmless and responded quickly to cessation of medication. They did not recur on resumption of medication. Yet they were rather upsetting for all the 5 patients and the one out-patient who developed a rash refused to continue with the drug. The excretion in the urine of a reducing substance which was not glucose did not give rise to signs or symptoms. It was, unfortunately, not possible to identify the substance with the means available in the hospital.

SUMMARY

The clinical trial on 51 patients described in this report suggests that Prep. 16038 (Ciba) in doses of 1,000 mg. or more per day, can be used with a moderate expectation of success in epileptic patients who have few seizures and who have EEG recordings with few paroxysmal signs. Patients whose epileptic state is labile as shown by large numbers of seizures in spite of complex and as far as one can say, maximal medication with anticonvulsant drugs and by EEG recordings with numerous paroxysmal signs did not respond well to doses of 1,000 to 1,500 mg. per day. Amounts of less than 1,000 mg. per day seem to have little effect.

Although in a number of patients Prep. 16038 was given alone, in one of them suppressing seizures completely, combination of the drug with other anticonvulsants would seem to be the method of administration to be recommended.

Unpleasant but harmless skin eruptions were observed in 5 patients. Two patients developed status epilepticus.

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HUSBANDS AND WIVES ADMITTED TO MENTAL HOSPITAL

By

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THERE have been a few studies of the nature and frequency of mental disorder in the offspring of marriages in which both parents were known to have been mentally ill (Schulz, 1940; 1941, Elsaesser, 1952; Lewis, 1957). A number of psychoses affecting both husband and wife have also been described as examples of folie à deux. Gralnick (1942) reviewed the literature, and found altogether 118 cases of folie à deux, but Ascher (1949) has concluded that such cases are commoner than would appear. The majority of those reported have involved blood relatives, in whom the implications of a "psychosis of association" are questionable, because of similarities in their genetic endowment (Kallmann and Mickey, 1946). However, Gralnick recorded 26 cases of folie à deux involving husband and wife.

Mental disorders in husband and wife which do *not* involve shared delusions (as in the case of folie à deux) would appear to be much more common, but their nature and frequency have not been studied adequately. Such information, however, is of great interest in relation to the possibility of assortative mating in certain types of mental disorder. Now, it is known that there are significant degrees of likeness between husband and wife with respect to many physical characteristics, and also intelligence. The study of assortative mating is important in any disorder which may be at least partly genetically determined, and is essential in planning preventive measures (Price and Halperin. 1940).

The present author has found only one previous study comparing observed and expected frequencies of mental disorders in husband and wife, and this was conducted by Penrose (1944) at the same mental hospital as the present investigation.

MATERIALS, METHODS AND RESULTS

During a period of four years commencing in July, 1954, the present author was senior psychiatrist on the female service at the Ontario Hospital, London, Canada, and kept track of husbands and wives admitted to this hospital. Eleven such couples were discovered, and particulars concerning each of these patients are recorded in Table I. Both members of eight of these couples (Nos. 4 to 11) were *first* admitted during the four-year period 1954–1957.

During this time the Ontario Hospital at London was the only public provincial mental hospital accepting patients from eight counties of Western Ontario (the counties of Bruce, Dufferin, Grey, Huron, Middlesex, Perth, Waterloo and Wellington). Figures obtained from the Dominion Bureau of Statistics (1958) indicate that in 1951 the total numbers of married males and of married females living in these eight counties each slightly exceeded 138,500.

TABLE I

Husbands and Wives Admitted to the Ontario Hospital, London, Canada, 1954–1957

	Sex	Year of Birth	Year of Marriage	Date(s) of Admission(s) to Ontario Hospital	Diagnosis	Treatment	Date(s) of Leaving Hospital
1	M	1918	1944	1. July, 1952	Paranoid schizophrenia	E.C.T.	December, 1952
				2. November, 1954	Psychosis with C.N.S. disease	Surgery attempted	Died May, 1956
	F	*1926*		*March, 1955*	*Reactive depression*	*Psychotherapy*	*April, 1955*
2	M	1926	1949	1. August, 1952	Paranoid schizophrenia	E.C.T.	November, 1952
				2. April, 1954	Paranoid schizophrenia	E.C.T. and insulin coma	June, 1954
	F	*1929*		*August, 1957*	*Schizo-affective psychosis*	*Chlorpromazine and psychotherapy*	*November, 1957*
3	M	1916	1945	1. December, 1952	Paranoid schizophrenia	E.C.T.	February, 1953
				2. March, 1954	Paranoid schizophrenia	E.C.T. and insulin coma	Still in hospital*
	F	*1922*		*December, 1954*	*Paranoid schizophrenia*	*E.C.T.*	*May, 1955*
4	M	1919	1945	1. June, 1954	Psychopathic personality	Returned to court	July. 1954
				2. August, 1954	Acute alcoholic psychosis	Detoxication	September, 1954
	F	*1928*		*July, 1956*	*Schizo-affective psychosis*	*E.C.T. and chlorpromazine*	*October, 1956*
5	M	1894	1922	December, 1954	Reactive depression and duodenal ulcer	E.C.T. and gastrectomy	April, 1955
	F	*1899*		*February, 1955*	*Manic depressive, depressed*	*E.C.T.*	*July, 1955*
6	M	1880	1917	December, 1955	Psychosis with cerebral arteriosclerosis	Custodial	Died May, 1956
	F	*1892*		*April, 1956*	*Psychoneurosis (mixed) and hypertension*	*E.C.T., drugs and psychotherapy*	*Still in hospital**
7	M	1888	1918	1. April, 1956	Chronic alcoholism	Psychotherapy	May, 1956
				2. September, 1956	Chronic alcoholism	Custodial	Still in hospital*
	F	*1899*		*June, 1957*	*Reactive depression (and alcoholism)*	*Psychotherapy*	*July, 1957*
8	M	1896	1946	May, 1956	Schizophrenia (type unspecified)	Custodial	Still in hospital*
	F	*1907*		*May, 1956*	*Paranoid schizophrenia*	*E.C.T. and ataractic drugs*	*Still in hospital**
9	M	1898	1923	1. October, 1945	Manic depressive, depressed	E.C.T.	December, 1945
				2. November, 1956	Manic depressive, depressed	E.C.T.	February, 1957
	F	*1901*		*January, 1957*	*Manic depressive, depressed*	*E.C.T.*	*March, 1957*
10	M	1902	1921	June, 1957	Without psychosis	Returned to court	July, 1957
	F	*1905*		*1. June, 1957*	*Without psychosis*	*Returned to court*	*July, 1957*
				2. April, 1958	*Paranoid state*	*E.C.T. and drugs*	*June, 1958*
11	M	1887	1921	August, 1957	Senile psychosis	Custodial	Still in hospital*
	F	*1897*		*June, 1957*	*Reactive depression*	*E.C.T.*	*August, 1957*

* Still in hospital, 30 June, 1958.

The total numbers of married couples living in this area during the period 1954–1957 is, therefore, estimated as about 135,000. Now annual reports of the Ontario Hospital, London, during these years showed an average annual intake of approximately 89 married male, and 86 married female, first admissions. *The random chance that both members of a married couple would be among the first admissions to the Hospital within the same calendar year* is, therefore 89×86÷135,000, which is 0·057 (or approximately 1 in 18). These figures are given in Table II, which also shows that the observed frequency was 0·5 or 0·75 per year, since during the four-year period, there were 2 mentally ill couples who were each first admitted within the same year (Nos. 8 and 11), and a third couple both diagnosed "without psychosis" (No. 10). The observed frequency was, therefore, at least 9 times as great as that expected on the basis of chance. Now the *expected frequency with which both members of a married couple would be among the first admissions to the Hospital during the entire four-year period* is 16 times as great as the expected frequency that both would be admitted in a single calendar year. This means that approximately 1 couple would be expected during the period 1954–1957, whereas the observed frequency was 8 couples.

TABLE II

Average Numbers of Husbands and Wives First *Admitted to the Ontario Hospital, London, Canada, During* Each *Calendar Year*, 1954–1957

	HUSBANDS	
WIVES	General Population	First Admitted to Hospital
General population	135,000	89
First admitted to hospital	86	0·75*

* If couple No. 10 is excluded on the grounds that both husband and wife were diagnosed "without psychosis" the observed average annual numbers of couples first admitted *during each calendar year* is 0·50. The expected number in either case is 0·057.

The observed number of couples first admitted *during the whole four year period* 1954–1957 is 8, while the expected number in this case is 0·9.

TABLE III

Frequency of Suicide or Admission to Mental Hospital Among Parents of 1,000 Patients Admitted to the Ontario Hospital, London, Canada, 1954–1958

	FATHERS		
MOTHERS	No History of Suicide or Admission to Mental Hospital	Committed Suicide or Admitted to Mental Hospital	Total
No history of suicide or admission to mental hospital	916	36	952
Committed suicide or admitted to mental hospital	46	2*	48
Total	962	38	1,000

* The expected number is 1·8.

The latter figures may be compared with those obtained by examining the *recorded frequencies of suicide or admission to mental hospital, among the parents of 1,000 patients* born during the four-year period, July, 1954 to June, 1958. This information was obtained as part of a more extensive study of familial data on these patients (Gregory, 1959). Among these 1,000 patients, 38 had a father, and 48 a mother, who was recorded as having either committed suicide or having been admitted to mental hospital. The number of patients expected to have had both parents thus affected is therefore $38 \times 48 \div 1{,}000$, which is approximately 1·8. These figures are given in Table III, which also shows that the observed number of patients with both parents thus affected was 2.

DISCUSSION

Penrose (1944) searched the files of the Ontario Hospital, London, and found 22 husbands and wives who had both been admitted since the beginning of 1910. Over the ten-year period immediately preceding his study, he found 4 couples first admitted to hospital within the same year (and a fifth couple who were both mentally defective), a frequency that was at least 9 times as high as random expectation. This corresponds very closely with the excess of observed over expected frequency of first admission of both partners within a given year (or over the entire four-year period) in the present study.

Penrose also recorded rates of mental illness found in parents of two large series of patients (1,280 patients with mental defect, and 1,000 patients with mental disease), and in each of these series found the observed frequency with which both parents were affected approximately 3 times as great as that expected on the basis of frequencies with which a single parent was affected. In the present study the observed frequency with which both parents were affected corresponded closely with the expected frequency. The numbers of affected parents in each of these three series is relatively small, and the discrepancy between these results, and those obtained by direct observation of husbands and wives both admitted to mental hospital, may possibly be due to chance, or to incomplete ascertainment of patients having both parents affected.

Re-examination of Table I shows that 7 of the couples here recorded had *similar diagnoses*—schizophrenia in Nos. 2, 3 and 8, depression in Nos. 5 and 9, alcoholism in No. 7, and "without psychosis" in No. 10. In at least certain of the remaining cases with dissimilar diagnoses, it appears that the wife's admission to hospital was directly related to her husband's illness (e.g. Nos. 1 and 11).

The *difference in the ages of husband and wife* at the time of their first admission to hospital was in all cases less than twelve years. In 9 of the couples, the *wife was younger* than the husband at the time of first admission, but in 8 cases the *husband was admitted first* (two couples arriving on the same day, and the wife being admitted first in only one instance).

In Gralnick's (1942) reviewed cases of folie à deux affecting husband and wife, the latter was the dominant partner in 15 out of 26 cases, and Wolff (1957) noted 7 further cases reported, in 6 of which the wife was the dominant partner. In spite of similarities in diagnosis, age at first admission, and time of admission, however, none of the couples in the present study satisfied the criteria for a supplementary diagnosis of folie à deux, with the possible exception of No. 10. In this case the wife subsequently turned out to be paranoid, and the husband, although not certified at this time and denying acceptance of his wife's delusions, impressed the wife's psychiatrist as probably sharing the latter's beliefs, at least to some extent.

The main findings of the present study appear to corroborate those of Penrose (1954) to the effect that both members of married couples are more likely to be admitted to hospital than would be expected, and that there is frequently a strong likeness in the type of disorder developing in husband and wife. He considered three possible explanations of these findings (*i*) that the husband and wife, living together, tended to influence one another and that one partner may "infect" the other with abnormal mental reactions, (*ii*) that similarity of environment, diet, economic state and common sources of anxiety, may contribute to common breakdown, or (*iii*) that there is a tendency for persons of like constitutions to marry one another. To these possibilities, the present author feels obliged to add (*iv*) that the mental illness leading to admission to mental hospital of one partner may precipitate a dissimilar mental disorder which leads to the admission of the second partner, (*v*) that if one partner comes to the attention of medical or social authorities and is admitted to mental hospital, pre-existing abnormality is more apt to be noticed in the other partner, who is therefore also more likely to be admitted to hospital within a short period of time. This last supposition is in keeping with observations that rates of admission to mental hospital do not simply reflect the frequency of mental disorders, but also a variety of complex socio-economic and administrative factors (e.g. Svendsen, quoted by Strömgren, 1950; Kramer *et al.*, 1955; Gregory, 1956).

Probably all these explanations are partly correct, and it may be impossible to assign them relative degrees of importance, even in individual cases. However, observed similarities in diagnosis of husband and wives admitted at widely different times, suggest that the first three factors listed are of some significance, and at least part of the undue frequency of mental disorder in husbands and wives may be attributable to assortative mating in certain of these disorders.

SUMMARY

The nature and frequency of mental disorders affecting both husband and wife are relevant to the possibility of assortative mating with respect to certain of these disorders.

Eleven couples admitted to the Ontario Hospital, London, Canada, during the past few years, have been studied, and the results compared with those of a similar investigation by Penrose (1944).

It is concluded that a variety of factors may be responsible for the unexpectedly high frequency of admission of both marital partners to mental hospital. Observed similarities in diagnosis of spouses admitted at different times, however, suggest a true increase in the frequency of similar disorders in the spouses of certain patients, which may be at least partly attributable to assortative mating among those predisposed to these disorders.

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MODELS FOR PREDICTION PURPOSES

By

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It is natural for psychiatrists and others dealing with mental patients to enquire what the prognosis is for a patient showing certain symptoms or syndromes of symptoms—taking age and the history of the patient's illness into account. The very fact that the symptoms generally form syndromes points to their interdependence to a greater or lesser degree, but this interdependence may not be a straightforward linear relationship such as is revealed by an ordinary correlation coefficient for the symptoms may interact with each other in a complicated way. When this is the case the problem of prediction is difficult from a statistical as well as from a psychiatric point of view. In this paper an effort is made to clarify the statistical problem by describing briefly and by comparing two statistical models which may be used for predictive purposes. The models are introduced by means of an example using data for patients who have attended this hospital. For reasons of clarity the number of variables included in the study is a bare minimum.

The Correlation Model

For a sample of 819 psychiatric patients, described in some detail in an earlier article in this journal (Trouton and Maxwell, 1956), the presence or absence of the three symptoms:

d	depressed
a	anxious
g	delusions of guilt

were noted. Two-by-two contingency tables for each pair of symptoms were drawn up and the tetrachoric correlations between the symptoms obtained. These were:

	d	*a*	*g*
d	—	0·385	0·600
a	0·385	—	0·289
g	0·600	0·289	—

The tetrachoric correlations between each symptom and the criterion, "recovered or much improved at time of discharge", which we will denote by the letter "c", were also found. They are:

	d	*a*	*g*
c	0·318	0·206	0·278

In an effort to assess the relative effects of the symptoms as regards recovery the customary approach is to employ a multiple regression model. Using it regression weights, known as partial regression coefficients, are calculated. These are the optimal weights to apply to the independent variables (in our case the symptoms) to obtain the highest possible correlation—the multiple correlation—between the independent variables and the criterion. These regression weights then give us a measure of the relative importance of the independent variables for predicting the criterion. For our data the regression coefficients with their standard errors are:

Variable	Regression Weight	Statistical Error	Significance Level
d	0·206	0·04276	$P<\cdot001$
a	0·089	0·03572	$P<\cdot02$
g	0·128	0·04122	$P<\cdot01$

The method by which they are obtained is given in most textbooks on general statistics.

Each of the regression weights is seen to be significant and the order of their importance for predicting the patients' recovery is, 1st—depression, 2nd—delusions of guilt, and 3rd—anxious. If then a patient is *depressed* his prognosis is slightly better than if he is *anxious*, etc. But what does the model tell us about the odds in his favour? The multiple regression coefficient throws some light on this problem. Each of the three symptoms under discussion is correlated only to a small degree with the criterion—the largest of the three correlations is 0·318. However, the multiple correlation between the three combined when the regression, or best weights, are used is 0·346, which is somewhat larger than 0·318. If the multiple correlation coefficient is squared and multiplied by 100 the figure 11·9 is obtained, and this is the percentage of the variance of the criterion which we can predict from a knowledge of the individual symptoms. This immediately tells us that any prediction we make about a patient's recovery from the presence or absence of these three symptoms only is unlikely to be very accurate. To improve our prediction we would require to take many more symptoms into account. Indeed apart from the labour concerned there is no reason why all 44 items in the earlier study (Trouton and Maxwell, 1956) might not be included in a prediction equation.

At this stage the reader may have noted an apparent contradiction. When the multiple correlation coefficient was squared and multiplied by 100 we obtained 11·9, and stated that we could, using all three symptoms, predict 11·9 per cent. of the variance of the criterion. Had we used only one symptom, say "d", which correlates to the extent of 0·318 with the criterion and had we squared this correlation and multiplied the answer by 100 we would have obtained 10·1, so that using just this one symptom we would claim to predict a smaller amount of the variance of the criterion than when we used three symptoms. It might appear then that a patient with just this symptom has a poorer prognosis than a patient with all three symptoms, but unless the symptoms interact (Meehl, 1950) to the patient's advantage such a conclusion would be absurd. The dilemma is cleared up if we note that regression weights are not independent of the units used when making our original observations and the regression weights given above refer to a scoring system where the variances of the independent variables are equal and the mean score of each is zero. For example if the presence of a symptom is denoted by -1 then the absence of that symptom should be denoted by $+1$, so that the mean is zero. If now we write out our regression equation as:

$$_{c}=0\cdot206d+0\cdot089a+0\cdot128g$$

where $_{c}$ stands for our estimate of c, and we compare the c-scores of two patients one of whom has all three symptoms while the other has only the second, then the first man's c-score is found to be $-0\cdot423$, while the second man's score is much higher, namely $0\cdot245$. We see now that whereas the amount of the variance of the criterion we can predict depends on the number of symptoms we take into consideration—in general the more the better—a man's actual predicted score on the criterion increases the fewer symptoms he has.

A Factorial Model

Tetrachoric correlations, as we have noted, are calculated from 2×2 contingency tables, but there is an underlying assumption that each pair of variables—though dichotomized—is distributed in a bivariate normal manner. It is known that even slight departure from normality causes the tetrachoric correlations to be extremely unreliable (Kendall, 1949), so that such correlations are not very trustworthy. With a multivariate model, like the multiple regression model, chances of error creeping in by way of tetrachoric correlations are even greater. To avoid the difficulty a more accurate method of dealing with multivariate dichotomous data has been outlined by Dyke and Patterson (1952), and others, and it will be well to look at our data in the light of their recommendations.

The presence (−) or absence (+) of the three symptoms for the 819 patients under consideration can be arranged as in Table I and the proportion of patients in each of the eight resulting categories that recovered can be examined.

Table I

Incidence of Symptoms Amongst Psychiatric Patients

Patients	819							
Depressed (d) ..	345 (+)				474 (−)			
Anxious (a) ..	211 (+)		134 (−)		172 (+)		302 (−)	
Delusions (g) ..	205 (+)	6 (−)	128 (+)	6 (−)	139 (+)	33 (−)	216 (+)	86 (−)
Recovered ..	68/205	3/6	58/128	3/6	70/139	23/33	129/216	59/86
Proportion recovered ..	0·3317	0·5000	0·4531	0·5000	0·5036	0·6970	0·5972	0·6860

The data when so arranged call to mind a 2^n factorial design in analysis of variance (Maxwell, 1958), and this suggests how they may be analysed.

A major disadvantage of correlational analyses, as Dyke and Patterson emphasize, is that they do not provide us with estimates of the effects of the separate factors—in our case the separate symptoms—involved in a study. To overcome this difficulty these writers prefer a model in which the effects of the separate factors may safely be taken to be additive and one from which accurate estimates of these effects and of their error variances may be obtained. In brief they want to be able to look at the proportion of patients in each of the eight categories (the last row of Table I) and to say what contribution the presence or absence of a symptom has made to it. Equally important they want to have a check on possible non-linear or interaction effects between the symptoms where recovery is concerned, and the multiple regression model—far from providing us with a test of the existence of such non-linear effects—tacitly assumes that they are either non-existent or unimportant. But let us return to the data in Table I.

Proportions, since by definition they lie between the limits 0 and 1, are not normally distributed. One of the consequences is that they cannot, without suitable transformation, be thought of as being composed of additive components. To put them on an additive scale Dyke and Patterson recommend a logit transformation, though a probit transformation would also have a lot to recommend it. Once the proportions have been transformed and put on an additive scale we can assume, as is the custom in analysis of variance designs, that each is composed of additive effects. In our example it will be convenient in the first place to assume that over and above the general mean of the eight transformed proportions there are three other effects—apart from random error—one due to each of the variables (or symptoms) being considered. It may well be that in addition to these there are other important effects due to interaction which should be considered but the method outlined by Dyke and Patterson furnishes a test of the possibility of the existence of such effects after the main ones have been taken into account.

An important additional point to notice is that whereas the readings in the cells of an ordinary factorial design are each based on the same number of observations, the numbers on which the eight proportions in our investigation are based differ widely. This fact greatly complicates the analysis of the data so that instead of a straightforward analysis of variance providing us with unbiased estimates of the several effects in which we are interested maximum likelihood estimates of these effects have to be obtained by an iterative process. The details of the method are outlined in detail by Dyke and Patterson so that only the results of the analysis of our data need be given here. If we denote by the symbol m′ the maximum likelihood estimate of the overall mean of the eight transformed proportions, and the maximum likelihood estimates of the effects of the three symptoms by the symbols d′, a′ and g′ then these estimates with their standard errors are found to be:

	Main Effect	Statistical Error
m′	·07827	·05338
d′	·19814	·03855
a′	·01030	·03708
g′	·13572	·05288

The estimate of the effect of d′ (depression) is highest, with that for g′ (delusions of guilt) second. These results agree with those found when the partial regression coefficients were obtained. The standard error of g′ is considerably larger than that for either d′ or a′. This was to be expected, for on examination of the frequencies in the fourth row of Table II, it is seen that for non-depressed patients the presence of the symptom "delusions of guilt" was very infrequent, only 12 cases out of 345 showing the symptom, so that an estimate of the effect of the symptom could hardly be expected to be very accurate.

Using the standard errors the estimates of each of the main effects can be compared with each other and with the estimate of the overall mean. The only significant difference obtained when these comparisons are performed is that between d′ and a′ ($P < \cdot 001$). This result bears out our earlier conclusion that prognosis for a patient suffering from depression is somewhat better than that for an anxious patient. The finding that none of the main effects differs significantly from the general mean is in keeping with the rather low correlations between the symptoms and recovery.

The final and perhaps most important stage in the present analysis is the test of the assumption that an additive model, which while allowing for main effects neglects interaction effects, is adequate for accounting for the variation in the data. The appropriate test is a chi-square test: for our data chi-square is found to be 6·639 which, with 4 degrees of freedom, is not significant. We may, therefore, rest assured that no interaction effects of note exist between the symptoms where recovery is concerned.

CONCLUSIONS

Two models, a multiple regression model and a factorial model, for use in prediction studies are illustrated by means of a practical example. The first model yields regression weights which indicate the relative importance of the independent variables for predicting the criterion, while the multiple correlation between the combined independent variables and the criterion furnishes a measure of the effectiveness of the battery as a whole for predicting the criterion. The model, however, has disadvantages in that it assumes a multivariate normal distribution for the variables concerned; it provides no check on possible non-linear or interaction effects between the variables nor does it furnish us with independent estimates of the separate effects of the variables. Moreover, in the present example, where the variables are dichotomous so that inefficient statistics such as tetrachoric correlations have to be used there is serious danger of error.

The factorial model, on the other hand, though laborious to use, allows—after a transformation of the data—independent estimates of the effects of the separate variables to be obtained together with estimates of their standard errors, and so makes tests of significance possible. Moreover, it provides us with a check on the existence of interaction effects between the variables when predicting the criterion.

For the sample of patients and the three variables, *depression*, *anxious*, and *delusions of guilt*, considered here it is found that there are no interaction effects where prediction of *recovery* is concerned. As a result we may expect the results given by each model to agree, though prior to a test for interaction we could not have asserted this with confidence. Of the three symptoms prognosis is slightly better for patients who suffer from *depression* alone than for those who suffer from *delusions of guilt*, or *anxiety*, in that order. Prediction on the basis of just these three symptoms, however, is not very reliable for the multiple regression model shows that together they can predict only 12 per cent. of the variance of the criterion.

To conclude it would appear that when multivariate dichotomous variables are concerned a multiple regression model is satisfactory if we are confident that the variables do not interact and if an examination of the 2×2 contingency tables convinces us the distributions are bivariate normal—a quick check on the latter condition is that entries in the diagonally opposite cells of the tables should be approximately equal. When these conditions are met the multiple regression model has the advantage that, though it involves laborious calculations when the number of independent variables is large, these calculations are less laborious than those involved in the *factorial* model. When, however, the assumption of normality is not adequately met and if interaction effects are suspected then the factorial model should be used.

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A correction to a previous article, by D. S. Trouton and A. E. Maxwell, is given overleaf.

A CORRECTION

The signs were omitted from Table V on page 18 of the article: "The Relation between Neurosis and Psychosis" (*J. Ment. Sci.*, 1956, **102,** 1–21). The corrected table is given below, by kind permission of the Editor-in-chief.

TABLE V

Orthogonal Rotated Factor Loadings

Variables	I	II	III	IV	V	VI	h^2
1	—·142	—·120	—·368	—·123	·118	·499	·448
2	·664	—·493	·176	—·003	—·215	·219	·809
3	·276	—·018	—·058	·056	—·134	·278	·178
4	—·029	·322	·022	·291	·051	—·124	·208
5	—·032	·349	·166	—·119	—·198	·120	·218
6	—·005	·468	·120	·001	—·203	·045	·276
7	·133	·382	·271	·159	—·178	—·302	·385
8	—·019	·308	·433	—·068	—·050	—·034	·291
9	·203	·276	·120	·461	—·114	—·178	·388
10	—·229	·247	—·105	—·237	—·035	·358	·310
11	·014	·483	·107	·097	—·220	—·176	·334
12	—·218	·539	·141	—·222	·095	—·061	·420
13	·120	·373	—·116	·149	—·089	·367	·332
14	—·108	·718	—·210	—·026	—·154	·017	·596
15	·084	·434	—·056	—·220	—·442	·133	·460
16	·019	·296	·389	·187	·068	—·029	·280
17	—·222	·425	·377	·007	—·167	·104	·410
18	·132	·157	·074	·018	—·385	·125	·212
19	·321	·093	·164	·095	—·040	·339	·264
20	·156	—·270	·008	·096	·131	·253	·188
21	—·130	·330	—·147	·037	·500	—·005	·399
22	·594	—·133	—·111	·435	·026	·143	·593
23	·397	—·126	·060	—·595	·101	·021	·542
24	·334	·180	—·350	—·308	·125	·132	·394
25	·556	·024	·202	—·516	—·130	—·081	·640
26	·615	—·002	·086	—·533	·050	—·186	·707
27	·315	·378	—·009	·263	·040	·405	·487
28	·612	—·148	·486	—·087	·139	—·033	·661
29	·302	·207	—·616	·355	—·113	·135	·671
30	—·016	·425	—·529	·031	·072	—·094	·476
31	·524	—·008	·372	—·381	—·006	—·169	·587
32	·587	—·004	·034	—·253	—·047	—·246	·472
33	·298	·224	—·031	—·351	—·201	—·214	·349
34	·396	·106	—·334	—·015	—·182	·196	·351
35	·625	—·463	·497	·031	—·014	—·201	·894
36	·539	—·040	·127	·020	·213	—·011	·354
37	·568	·104	—·490	·206	—·030	·057	·620
38	·573	—·275	·135	·088	·033	—·432	·618
39	·667	—·221	·125	—·059	·226	—·191	·600
40	·604	—·242	·254	—·135	·206	—·308	·643
41	·308	·192	—·431	—·176	·176	·339	·494
42	·389	—·010	—·086	·184	·208	·350	·358
43	—·040	·397	—·202	—·010	·532	·021	·484
44	·207	—·230	·579	—·284	—·259	·053	·582
45	—·032	—·163	—·580	·174	—·059	·082	·404

A. E. MAXWELL

SYMPTOM CLUSTERS AND PERSONALITY TYPES AMONG PSYCHONEUROTIC MEN COMPARED WITH WOMEN

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INTRODUCTION

IN a previous investigation (Foulds and Caine, 1958) a series of consecutive admissions (women) admitted to Runwell Hospital and diagnosed as psychoneurotic were found to divide into four groups in terms of presenting syndrome (hysteria or dysthymia) and of personality type (hysteroid or obsessive). When psychological tests were administered to these groups it was found that some measurements were related to diagnostic category whilst others were related to personality type.

The present study is concerned with the validity of these classifications in a group of male psychoneurotics. In addition, test measures and other data derived from the case notes are compared for neurotic men and women.

SUBJECTS AND THEIR CLASSIFICATION

The female group has been described in detail in the study mentioned· As a comparable male group 62 successive admissions to Runwell Hospital who were diagnosed as psychoneurotic were included in the present study. As with the female group, for the diagnostic classification the final psychiatric diagnosis—uncontaminated by test results—was accepted.

For the personality classification two psychiatrists independently rated the personality of the subjects on the five-point hysteroid-obsessive rating scale used in the earlier study. The combined results of the two ratings were then dichotomized. If the two failed to agree a third rater was used.

Classification comparisons of men and women were as follows:

	Men	Women
1. Inter-psychiatrist correlation	·66	·67
2. Percentage of subjects on whom two psychiatrists failed to agree regarding personality type	15½	14
3. Hysterics of hysteroid personality (Hh)	16	16
4. Hysterics of obsessive personality (Ho)	2	4
5. Dysthymics of hysteroid personality (Dh)	21	26
6. Dysthymics of obsessive personality (Do)	23	22

Diagnosis of the male subjects by psychiatrists seemed to present more difficulty than in the females. A greater variety of class names and of secondary features was used and the diagnosis tended to be changed rather more often before the final formulation.

Dysthymics were significantly older than Hysterics in both the male and female groups. In the case of the latter the diagnostic groups were equated for age and in the former Spearman rank-order correlations were calculated between age and the test measures within each of the three main group classifications. The male Hh group was so much younger than the other groups that age-matching would have resulted in a complete distortion of the sample. These correlations, together with the p values for one-tail tests, are shown in Table I for all test measures. None of the measures which systematically differentiated between groups was significantly related to age within groups.

TABLE I

Rank Order Correlations With Age

	M.H.V.	P.M.	Maze T.T.	D–Hs	Ex.	In.	D+Pt	S.I.	L.P.	P.M.T.	T.W.	Dis.
Hh	·04	−·09	·65†	−·02	·49*	·16	−·26	·64†	−·60†	·34	·11	−·32
Dh	·21	−·47*	·60†	−·20	·07	−·11	−·19	·12	·53†	·26	·37	−·07
Do	·18	−·25	·26	−·02	−·38*	−·03	−·18	·18	−·49*	−·12	·17	−·01

* p<·05. † p<·01.

From Table I it would appear unlikely that potentially useful measures for differentiating between groups have been obscured by any relationship with age. In some instances (e.g. Maze: Total Time) the correlations have worked against the differentiation and in others the correlations have gone in opposite directions. The only measure with three significant correlations was Mazes: Lifted Pencils (L.P.). The differences between groups were, however, so very small that matching for age would have made little difference.

THE TESTS

The tests and their administration were identical with those used with the female subjects, namely: the Mill Hill Vocabulary Scale, the Progressive Matrices (1938), the Porteus Mazes, the Thematic Apperception Test, the Minnesota Multiphasic Personality Inventory, the Superiority-Inferiority Index and the Tapping Test.

DATA DERIVED FROM CASE NOTES

These data included personal problems complained of by the patients or noted by relatives concerning the patients. Circumstances attending admission to hospital, accidents at work (usually compensation cases), previous physical illness, treatment in the hospital by E.C.T and outcome (as noted on the discharge form) were also tabulated from the notes for each subject by the authors.

RESULTS

With women subjects the test results tended to fall into three rather distinct groups: those which were not significantly different between any of the groups; those which were significantly different between diagnostic categories but not between personality types and conversely. With the males this was confirmed; but the test measures were somewhat different and the results somewhat less clear. Table II shows the differences between each pair of male groups for all

TABLE II

Mann Whitney U for $n_2 > 20$ (2 Tail)

	Hh and Dh		Hh and Do		Do and Dh	
	Z	p	Z	p	Z	p
M.M.P.I. D–Hs	0·12	·9044	1·64	·1010	1·62	·1052
M.M.P.I.: D+Pt	2·24	·0250	2·24	·0250	0·32	·7490
M.M.P.I. Ex:	1·33	·1836	0·07	·9442	1·56	·1188
M.M.P.I. In:	1·29	·1970	2·76	·0058	1·88	·0602
Maze distraction	0·09	·9282	1·84	·0658	1·72	·0854
Maze tracing time	1·01	·3124	2·10	·0358	1·37	·1706
Maze lifted pencils	1·13	·2584	0·58	·5620	1·79	·0734
P.M. score	0·23	·8180	0·01	·9920	0·38	·7040
P.M. time	1·72	·0854	3·83	·0001	2·64	·0082
Mill Hill Vocabulary	0·95	·3422	2·23	·0258	1·08	·2802
F/B: S.I.	0·86	·3898	1·30	·1936	1·00	·3174
T.A.T. total words	0·95	·3422	0·74	·4592	0·95	·3422
Tapping	0·40	·6892	0·05	·9602	0·36	·7188
Age	1·85	·0644	2·86	·0042	1·26	·2076

TABLE III

Significant t and p Values of Case Note Data in Relation to Diagnostic and Personality Classification

		Hh and Dh		Hh and Do		Dh and Do	
1. Friction with parents	F						
	M			5·633	·02	6·38	·02
2. Interpersonal conflicts (including 1 above)	F	7·90	·01	17·98	·001		
	M	4·069	·05			14·36	·001
3. Illness	F			4·97	·05	3·80	·10
	M	2·897	·10	3·765	·10		
4. Work	F						
	M	4·37	·05	6·81	·01		
5. E.C.T.	F			4·97	·05	6·50	·02
	M			8·43	·01	7·74	·01
6. Seven-day improvement	F					3·465	·10
	M			11·560	·001	5·376	·01
7. Recovered	F	5·06	·05	6·18	·02		
	M						
8. Accident	F						
	M	6·842	·01	7·516	·01		
9. Promiscuity and/or frigidity	F	2·297	·10	4·318	·05		
	M						

measures. Table III shows an analysis of data derived from the case notes in relation to diagnostic categories and personality types, for both males and females. The results of these tables are summarized, taking proximity to the 5 per cent. level as suggestive.

Psychological Tests (Median Scores, Italicized for Associated Groups)

Measures Related to Diagnosis

Males	Hh	Dh	Do	Females	Hh	Dh	Do
M.M.P.I. D+Pt	130	*177*	*162*	M.M.P.I. D—Hs	5	*12*	*13*
				Maze distraction	77	*66*	*68*
				T.A.T. Productivity	101	*78*	*84*
				Tapping	26	*4*	*5*

Measures Related to Personality Type

Males	Hh	Dh	Do
Matrices time (minutes)	*29*	*32*	48
Maze Distraction	*78*	*79*	63
M.M.P.I. Intropunitive Scale*	*7*	*9*	12

Females	Hh	Dh	Do
Matrices time (minutes)	*33*	*36*	56
Maze Lifted Pencils	*8*	*7*	16
Maze Total Time (seconds)	*250*	*274*	368
Superiority Index	*3*	*1*	−1
M.M.P.I. Hostility Scale	*9*	*9*	6

Measures Differentiating One Pair Only

Males	Hh	Dh	Do
Maze Lifted Pencils	3	2	4
Maze Total Time	234	278	289
Mill Hill Vocabulary	44	52	57

* A scale devised by the authors from the M.M.P.I. involving self-critical statements. A full description of the scale has been published (*J. Ment. Sci.*, **105**, 438, 1959).

Taking the optimal cutting point on the M.M.P.I. D+Pt diagnostic measure resulted in 69 per cent. of the male cases being correctly diagnosed as compared with 78 per cent. reported of the female group on their three diagnostic measures.

Taking the optimal cutting point on the personality measures of Matrices time, Maze distraction and M.M.P.I. Intropunitive scale for the men resulted in 74 per cent. of cases correctly classified as compared with 78 per cent. found with the females.

For the men 52 per cent. only of cases, as against 60 per cent. for the women, were in agreement with both the clinical diagnosis and the personality rating.

Data Derived from Case Notes (*Percentages, Italicized for Associated Groups*)

Related to Diagnosis

Males	Hh	Dh	Do
Dissatisfaction at work	19	*52*	*61*
Accidents	37½	*0*	*0*
Illness	6	*29*	*39*

Females	Hh	Dh	Do
Considered to have recovered	19	*54*	*59*
Promiscuity/Frigidity	50	*27*	*14*
Interpersonal conflicts	94	*54*	*36*

Related to Personality

Males	Hh	Dh	Do
Given E.C.T.	*0*	*9½*	48
Friction with parents	*6*	*9½*	43½

Females	Hh	Dh	Do
Given E.C.T.	*19*	*19*	55
Considered to have improved within 7 days of entering hospital	*50*	*54*	27
Illness	*19*	*27*	55

Not Systematically Related to Diagnosis or Personality

Males	Hh	Dh	Do
Interpersonal conflicts	56	90	35

In view of the fact that the case note data were originally collected in a relatively unsystematic way, it is surprising that such marked differences between the groups have been found. As with the psychological tests, the case note material differs between males and females and was associated with diagnostic and personality classifications.

These findings were as follows:

1. Hysteroid men tended to report trouble with their parents more frequently than obsessive men. The trend was the same, but not significantly so, among women.

2. Inter-personal conflicts were stressed by Dysthymic men of hysteroid personality, but by Hysteric women of hysteroid personality.

3. There was a non-significant tendency for male Hysterics to stress either their own or someone else's illness less as a precipitating factor in their illness and for obsessive Dysthymic women to do so more to a significant degree.

4. Personal failure on the job, such as inability to cope, letting their mates down, etc., was mentioned significantly more often by Dysthymic men (most of the women were not in employment outside the home), the fault was theirs rather than the type of employment. It is interesting to speculate on the significance of such attitudes for vocational guidance.

5. E.C.T. was given much more frequently to obsessive Dysthymics (and within this group to Depressives rather than Anxiety States).

6. Improvement was noted within the first week in hospital much more frequently among men of hysteroid personality; the trend was there for the women, but not to a significant degree. This observation was anticipated in the previous study.

7. Dysthymic women were more frequently judged to have recovered on discharge.

8. Accidents were mentioned as a precipitant only in the case of male hysteroid Hysterics.

9. Promiscuity and/or frigidity was commonest among hysteroid women. In fact, there was a tendency for hysteroid Hysterics to be promiscuous and for hysteroid Dysthymics to be frigid. The incidence of promiscuity or impotence among men was randomly distributed between the groups. Mention was made in the notes of such problems in one case in four for men and women.

Discussion

The value of a double classification involving diagnosis and personality type has been supported both by psychological tests and clinical data in the present study. There are, however, very considerable sex differences in the relevant test measures related to these phenomena and these too are reflected clinically. One of the outstanding differences shown by this study is that it is more difficult to distinguish between male than between female groups. Since the psychiatrists too found this task more difficult, it may be that within the neurotic population men are more homogeneous than women. On the other hand, it might be said that one of the effects of some hypothetical sex-bias in the psychiatrists is to diagnose dysthymia in men and hysteria in women with the same cluster of signs and symptoms in mind. Twenty-nine per cent. of both men and women were, in fact, diagnosed as Hysterics; 60 per cent. of men and 62 per cent. of women were rated as hysteroid in personality. This is not, of course, definitive evidence against the possible sex-bias; but, since no means exist for obtaining such evidence, this hypothesis cannot at present be entertained.

That sex differences are operative in two of the psychological tests used in the present study has been recognized previously. Allowance has been made for these in the T scores of the M.M.P.I., but since this test was standardized in the United States of America somewhat different norms might well appertain in this country. The indication here is that the adjustment has been insufficient for a British population, with a larger original discrepancy between men and women. Porteus (3) has reported sex differences in Maze performance: "sex differences seemed to cut across this (test)* situation, the better class boys having distinctly better average Maze performance than girls, this sex advantage being less marked for children of lower social grade". ". . . with girls temperamental defects have a greater relation to success in the tests than is the case with boys." Foulds (2) found that total tracing time was significantly different for men and women, with men working more quickly (male mean, 19·90, S.D. 6·90; female mean, 26·00, S.D. 11·96) and this sex difference was thought to carry over into neurotic performance. The attitude of the sexes may well be somewhat different towards this type of test material, with men being more task centred than women. This would account for Porteus' statement that temperamental defects affect girls' performance more than boys', an observation which is borne out in the present study.

Extrapunitive or intropunitive expression are related to personality rather than diagnostic category for both women and men respectively with obsessive women being markedly less extrapunitive than all other groups of both sexes and hysteroid men being markedly less intropunitive. The relationship between extrapunitive and intropunitive expression is an obscure one whether one accepts the Freudian dichotomy or not. A paper analysing the relation of M.M.P.I. scores on these two scales, in some detail, is in the course of preparation.

In any event, before simplification and refinement of these tests is attempted, further typical samples of male and female neurotics should be tested on the present battery to confirm these sex differences. In addition, final classification of the test measures in terms of diagnosis and personality should await the collection of a comparable group of hysterics of obsessive personality for both sexes. Without such a group the relationships are determined solely by the performance of the Dh's.

With regard to the clinical data the trends are in the expected direction. The association of certain personal problems with personality type rather than with diagnosis, however, may well have contributed to the unreliability of psychiatric diagnosis in the past. The clinical data, as presently analysed, do support the psychological tests in that the hysteroid man or woman with dysthymia behaves differently, has different attitudes and is treated differently from his obsessive counterpart. To attempt any theory of the relationship between psychiatric illness, personality type and environmental influence at this stage is obviously impossible and premature. Suffice it to say that rough preliminary classifications and some relevant observations have been made.

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* Present authors' brackets.

SUMMARY

A group of 62 male neurotics were classified as to diagnostic category and personality type and compared with regard to test measures and clinical data with a comparable group of female neurotics previously described. The value of such a double classification system was supported by both the test measures and the clinical data, but a large number of sex differences were found.

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DISULFIRAM AND CITRATED CALCIUM CARBIMIDE IN THE TREATMENT OF ALCOHOLISM

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I. DISULFIRAM

SINCE disulfiram (tetraethylthiuram disulphide) was first reported to cause intolerance to alcoholic beverages (Hald *et al.*, 1948) it has been used extensively as an adjunct in the treatment of alcoholism. Impressions of most therapists have been favourable (e.g. Martensen-Larsen, 1953; Wallerstein *et al.*, 1957). At Warlingham Park Hospital the drug has been administered over the past five years to more than 250 alcoholics (as part of a fairly comprehensive therapeutic programme—Glatt, 1955) and has been found by a great many patients very helpful after discharge from hospital.

However, disulfiram has a number of undesirable side-effects. Thus among 69 W.P.H. patients receiving the drug (1 gm. on the first night, followed by ½ gm. on each of the following nights) prior to a disulfiram-alcohol test, 28 occasionally complained of daytime fatigue, 24 of a sleepy feeling in the mornings, 17 of headache, 17 of a feeling of reduced vitality, 12 of bad taste in the mouth or unpleasant odour in the breath, 11 of dizziness, 10 of shortness of breath, and 9 of gastro-intestinal symptoms (chiefly nausea). Such side-effects were usually mild and disappeared almost always with the reduction of the amount taken, after the test, to a minimal maintenance dose (Martensen-Larsen, 1953). No case of provocation of psychotic reactions by disulfiram—as described for example by Martensen-Larsen (1951)—was seen among our patients.

Other drawbacks attending the use of disulfiram are caused by the unpredictability of the severity of the *disulfiram-alcohol reaction.* On the one hand a few of our patients hardly showed any response at all during this test (and of these, two later experienced a severe reaction when, having had disulfiram, they took drink some hours later while on leave from the hospital). On the other hand the reaction may be very severe and fatalities have been recorded in the literature following a disulfiram-alcohol reaction. Four "unexplained" deaths of this sort, after sudden respiratory or circulatory collapse, were found by Jacobsen (1952) in a review of the literature and of 11,000 Danish patients. The risk that certain alcoholics will take the gamble of drinking in spite of previous ingestion of disulfiram cannot be excluded, though in our experience alcoholics who intend to drink usually make sure of discontinuing disulfiram a few days beforehand.

In our own experience with 69 closely observed disulfiram-alcohol tests (Table I) reactions of all kinds of severity were seen but there was only one case of a fairly severe circulatory collapse. About 75 per cent. of these patients

TABLE I

Symptoms Frequently Seen During Disulfiram-Alcohol Test

	44 Males		25 Females	
Strength of Reaction	Slight	Severe	Slight	Severe
Flushing of face	21	23	17	8
Palpitations	15	20	12	4
Nausea	22	12	12	3
Universal flushing	21	3	12	4
Sleepiness: yawning	21	6	10	1
Headache	17	5	8	8
Constricted feeling (neck, chest)	24	4	6	2
Difficult breathing	22	5	7	1
Vomiting	15	9	5	2
Choking sensation	17	2	4	3
Dizziness	17	—	6	2
Excitement	11		9	
Depression	11		3	

had received the last disulfiram tablet prior to the test on the previous evening, 25 per cent. had the tablet on the morning of the test, i.e. two hours beforehand. The amount of alcohol given was two to four ounces of spirit, or one pint of beer. Eight patients underwent a second test about a week afterwards because the first test had hardly evoked any response. In most cases the reaction set in shortly after the alcoholic drink was taken but in nine males and in six female patients the onset of the reaction was delayed for more than three hours.

With the stated amounts of alcoholic drink given, the reaction appeared relatively more severe in the beer than in the spirit drinkers (Table II). Two wine drinkers had a "moderate" reaction.

TABLE II

Severity of Disulfiram-Alcohol Reaction in Beer and Spirit Drinkers

Strength of Reaction	54 Spirit Drinkers	13 Beer Drinkers
Mild	13	1
Moderate	21	5
Severe	20	7

In a comparison of the strength of reaction of those who had taken disulfiram for three, four, five and six nights respectively, prior to the test, contrary to our clinical impression the severity of the disulfiram-alcohol reaction was not greatest in those with the longest disulfiram "build-up" (Table III).

TABLE III

Severity of Disulfiram-Alcohol Reaction in Reference to Preceding Disulfiram "Build-Up"

Number of nights (prior to test) with disulfiram medication	3 Nights	4 Nights	5 Nights	6 Nights
Strength of Reaction	(7 Patients)	(32 Patients)	(14 Patients)	(14 Patients)
Mild	1	5	3	5
Moderate	3	13	7	3
Severe	3	14	4	6

II. CITRATED CALCIUM CARBIMIDE (TEMPOSIL)

At the Alcohol Research Foundation in Ontario a search has been undertaken in recent years for drugs which, like disulfiram, made the taking of alcohol unpleasant but would be free from untoward side-effects. Cyanamide (carbimide), a substance with a chemical constitution quite different from disulfiram, had been described as causing hypersensitivity to alcohol as long ago as 1914 (Koelsch, 1914) and, when used in industry, was known to exert an unpleasant effect on workers who took alcohol. The reaction was known industrially as "mal rouge" and was characterized by marked flushing of the face with severe headache, tachycardia and dyspnoea. Apart from the unpleasant reactions after drinking alcohol this compound had been found to have few toxic effects. A number of different carbimide derivatives were evaluated (Ferguson, 1956), and recently Armstrong and Kerr (1956) and Bell (1956) reported on the use of a citrated calcium carbimide ("Temposil"). They found that this compound, in 50 mg. doses, will sensitize an individual to alcohol. Up to the present time they have found little evidence of toxicity among patients who have been placed on this drug for long periods. Armstrong (1957) in a review article on protective drugs in the treatment of alcoholism states that he had at least 29 patients on citrated calcium carbimide for periods of three to nine months without reporting symptoms. However, an elevation of the white cell count was common.* No deaths have been reported as a result of reactions with calcium carbimide nor have there been any reports of psychosis in patients receiving the drug.

In comparing the use of disulfiram and calcium carbimide he states that with calcium carbimide the patient becomes sensitive to alcohol within a matter of hours whereas with disulfiram it often requires a matter of days of medication to obtain an adequate reaction. However a patient on disulfiram can have a reaction for three to four days and sometimes as long as 10 days after stopping therapy whereas with calcium carbimide it is unlikely that there will be any reaction to alcohol once the drug has been discontinued for three days. The reaction of patients on calcium carbimide who take alcohol is not so severe as with disulfiram, and it is Armstrong's impression that a somewhat larger number of patients fail to have a reaction at all with alcohol when on calcium carbimide. At present the dosage recommended by Armstrong is 100 mg. daily as compared with 500 or 250 mg. of disulfiram.

J. A. Smith *et al.* (1957) in a study of 73 male alcoholics who were given citrated calcium carbimide claim that a single dose of 50 mg. of the drug sensitizes an individual to alcohol. They did not observe with the new drug the marked hypotensive effects frequently seen in a disulfiram-alcohol reaction. It appeared from their study with calcium carbimide that it has no undesirable side-effects such as lethargy, impotence and abdominal discomfort which are complained of by some patients who take disulfiram. It appeared also that calcium carbimide has to be taken daily to be effective since it was much more rapidly excreted than disulfiram.

Present Investigation

Forty-one alcoholic in-patients at W.P.H. were studied as regards the effects of the administration of citrated calcium carbimide when (*a*) taken by itself and when (*b*) followed by the taking of alcohol. The dosage varied from 50 mg. to 100 mg. (Temposil) daily.

(*a*) No patient complained of bad breath or bad taste in the mouth while on the drug. Five patients complained of itchiness of the palms of the hands; four of skin irritation, which also included itchiness of palms of hands and feet; two patients had an erythematous skin rash; one complained of gastric upset on the second day after taking the drug; three others complained of reduced vitality. All these complaints were mild and transient, and studies of blood and urine, liver function tests and electrocardiograms did not yield any abnormal findings.

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* Subsequent studies did not confirm this finding (Armstrong, J. D., *Canad., M.A.J.*, 1958, **79**, 292).

(*b*) *Citrated calcium carbimide—alcohol test.* This test was carried out in some patients who had received the drug for several days beforehand, in others on the first day of taking Temposil.

On the day of the test the patients were given 50 mg. or 100 mg. of Temposil one hour before the test, usually on an empty stomach. An hour later they were given an alcoholic drink, not more than the equivalent of 2 ounces of 70 proof whisky. In most patients a reaction took place within five minutes of taking the drink. There was an increase in the pulse rate and respiratory rate, generalized flushing of the face with conjunctival injection, tremors of the hands, occasionally pain in the chest, severe frontal headache, nausea but rarely vomiting. There was little alteration in the blood pressure. These signs and symptoms usually reached a maximum after 30 minutes, after which they started to abate. The reactions were usually over within an hour and a half. The strength of the reaction did not seem greater in those patients who had been given Temposil for several days beforehand than in those who had taken it for the first time on the day of the test.

Of the 41 patients on Temposil two showed no reaction to alcohol.

Among laboratory tests carried out during and after the Temposil-alcohol test the only abnormality observed was a transient moderate leucocytosis (up to 20,000/cmm.) arising during the test in several patients and subsiding within 24 to 48 hours.

At the present time a number of patients are on maintenance doses of Temposil (50 mg.) and so far no adverse reactions have been reported.

DISCUSSION

The mode of action of citrated calcium carbimide is said to be dependent on the inhibition of one or more enzymes which are required to oxidize acetaldehyde. It may have an additional property of altering the vascular reaction to acetaldehyde. As with disulfiram alcohol metabolism is slowed down by the inhibition of these enzymes so that acetaldehyde accumulates in the blood, reaching levels as high as ten times that which are normally found during the breakdown of alcohol. The presence of this large quantity of acetaldehyde in the blood produces a number of toxic symptoms such as flushing of the face and body, headache, dyspnoea, palpitations, nausea and occasional vomiting.

Of six patients who had previously also undergone a disulfiram-alcohol test only one experienced a stronger reaction with the Temposil-alcohol test. That on the whole the Temposil-alcohol reactions are much less severe than disulfiram-alcohol reactions may not be an unmixed blessing. In one of our patients the Temposil-alcohol reaction was so mild as to allow the reactivation of his "craving" by the given alcohol test dose, so that he commenced drinking alcohol again and it took some time to get him under control.

As to the deterrent effect of a discomforting test reaction it has been pointed out by Smith *et al.* that the discomfort of such reactions may in itself be of little therapeutic value since alcoholics frequently undergo very unpleasant experiences after drinking bouts without marked influence on their drinking pattern; but the fact that the unpleasant reaction follows almost immediately the alcohol intake seems to have exerted a very salutary deterrent effect in the past on our patients who were on a disulfiram maintenance therapy.

In our small series of cases the incidence of side-effects with Temposil was higher than previously reported, but nevertheless side-effects with Temposil seem to be less frequent and much milder than with disulfiram.

In view of the relative mildness of the Temposil-alcohol reaction it seems to be risky to carry out such a test in an out-patient clinic as the alcohol test dose may reactivate "craving". Should the reaction have been so mild that the patient himself feels it might not deter him from drinking in the future, it might be preferable to carry out a subsequent disulfiram-alcohol test and to suggest maintenance disulfiram maintenance treatment. In view of the relative unpredictability of the reactions to disulfiram-alcohol and Temposil-alcohol tests, many—but by no means all—alcoholics will not take a drink having taken either disulfiram or Temposil, once they have been warned about the consequences or having watched the test reactions in other patients. In such cases it might be possible to suggest regular medication with the milder Temposil. Likewise Temposil may be preferred for those patients in whom it is feared that they might gamble on getting away with a few drinks in spite of having taken a sensitizing drug, but it is more than doubtful whether such individuals will do well with any such drug. Temposil may also be indicated in the very rare cases with persistent side-effects with even a reduced disulfiram-maintenance dose, and in those alcoholics whose disulfiram-alcohol reaction had been very severe. Finally the choice of the drug depends on an assessment of the individual patient's personality: some alcoholics feel "certain" that they would not risk even a mild reaction (such as the one produced by Temposil), others do not trust themselves in that respect unless there is a likelihood of a stronger reaction (such as the one often following disulfiram).

Clearly the value of Temposil cannot yet be finally estimated. Moreover one should not lose sight of the fact that both disulfiram and Temposil are no more than auxiliary weapons in the overall treatment of alcoholism. Thus most patients apart from physical therapies also require some form of psychotherapy and derive invaluable benefit from linking up with Alcoholics Anonymous.

Conclusion

Disulfiram is a valuable adjunct in the treatment of alcoholism but has certain drawbacks. A new drug, citrated calcium carbimide, has therefore been tested in a number of alcoholics. Its final value cannot yet be estimated but the preliminary experiences seem to warrant further trials.

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TRANQUILLIZERS, A LATIN SQUARE TRIAL

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This study is an attempt to assess the value of certain drugs commonly used in psychiatric practice in decreasing the hyperactivity of patients. It is one of a series investigating tranquillizers and mental disorder in mental deficiency practice (Craft 1958).

Six drugs were used. Amylobarbitone was included as a sedative whose action was well known. Amphetamine is said by Bradley (1950) to be effective in behaviour disturbances associated with epilepsy, whilst promazine has been found effectively to sedate aggressive epileptic defectives by Bergin and Bergin (1958). The sedative properties of promethazine have recently been noted (Erwin, 1957) and encouraging reports of reduction of hyperactivity by prochlorperazine have been given (Lecomte *et al.*, 1957). Acetyl promazine, the sixth drug used, has been found of distinct value by Collard and Maggs (1958) in disturbed schizophrenic patients.

A Latin square trial with tranquillizers has previously been reported by Raymond *et al.* (1957) who based his study on work by Williams (1949). Raymond compared a placebo with amylobarbitone, meprobamate, chlorpromazine, benactyzine and sedaltine and found amylobarbitone significantly superior to the others in terms of patients' assessment of their own improvement.

Although the design of the present study was based on that of the foregoing authors, there were important differences. The present trial involved 18 patients only, and they were mental defectives. The patients were selected on account of their outstanding hyperactivity, aggression and socially bad habits. They had needed tranquillizers during the previous winter and the rating scales developed in preceding trials were used again (Craft, 1957–8). These scales consisted of daily ratings by nurses on three dimensions; activity, graded from destructive of any article (+6), through normal (0) to stuporose (−6); aggression, ranging from actual injury to others (+6), through normal (0) to actual injury to self (−6); and social behaviour, spitting and shouting (+6) through normal to (0) to eating excreta (−6). This trial was an attempt to assess the true taming or tranquillizing effect of these drugs, for one wished to see the hyperactivity reduced without excessive sleep inducement, and both injury to others and to self reduced.

Clinically the majority of patients appeared brain injured, ten being epileptics, although three had schizophrenia. All patients were below I.Q. 34, some being imbeciles, the majority being high grade idiots. Ages ranged from 15 to 44, average 25·2 years old.

The Chief Pharmacist of the hospital ensured that the administration of drugs would follow the design set out below, and that the experiment was blind from the medical and nursing point of view. Despite the tablets looking similar the nursing staff knew from previous trials that it was essential for patients to take their individually labelled tablets. The experiment was spread over a period of 165 days, being a preliminary period of 39 days followed by 6 equal periods of 21 days, during each of which one of the drugs was administered. It was felt that a period of a fortnight on each drug, as used by Raymond, was too short for an adequate assessment, for many of the tranquillizing drugs need time for effective action.

During the first week of each three-week period a stepwise increase in dosage of drugs was arranged by the pharmacist so that one tablet was taken the first and six on the final day. A similar stepwise decrease occurred during the third week. During the second week each patient took six tablets of the appropriate drug daily, i.e. amylobarbitone gr. 6; amphetamine sulphate 30 mg.; promethazine hydrochloride 150 mg.; acetyl promazine 150 mg.; promazine hydrochloride 600 mg.; and prochlorperazine 150 mg.

Of the 18 patients involved in this experiment, 6 were from one ward and 12 from the adjacent ward. These were divided into three groups of 6. In each group of six the drugs were administered according to the following experimental design, each symbol standing for a drug.

Table I

	Three Week Period					
	1	2	3	4	5	6
Patient 1	A	C	B	E	F	D
Patient 2	B	D	C	F	A	E
Patient 3	C	E	D	A	B	F
Patient 4	D	F	E	B	C	A
Patient 5	E	A	F	C	D	B
Patient 6	F	B	A	D	E	C

This design ensured that each patient received each drug during one period of the experiment, and that during any period all 6 patients in any group were receiving a different drug. Also each drug is followed by every other drug once only in each group. Following Williams (1949) this enables the residual effect to be estimated, that is the effect of the drug after it has been discontinued. In fact the analysis here mainly follows that suggested by Williams.

During the whole period of 165 days the behaviour of the patients was recorded on three counts; there being no losses of patients or of observations during the trial. Since the purpose of the tranquillizers is to make a patient more normal, the absolute value of any observation can be used in the assessment of drug action, i.e. ignoring plus or minus signs. These can be taken into account if more detailed analysis is required later.

Results

The average behaviour for patients on each drug are given in Table IIa, Table IIb showing the residual effects adjusted for treatment effects. It should

Table IIa

Average Behaviour Away from Normal on Rating Scales

Average for All Patients	No Drug	Amylo-barb.	Amphet-amine	Pro-methazine	Acetyl Pro-mazine	Pro-mazine HC1	Prochlor-perazine
Aggression	0·67	1·03	1·16	0·80	1·36	1·27	1·01
Activity ..	2·21	2·82	2·50	2·53	2·42	2·18	1·68
Social behaviour	2·50	3·21	2·78	3·16	2·76	2·89	2·51

TABLE IIb

Average Behaviour Away from Normal on Rating Scales

Residual Effects (adjusted for treatment effects)	Amylo-barb.	Amphet-amine	Pro-methazine HCl	Acetyl Pro-mazine	Pro-mazine HCl	Prochlor-perazine
Aggression	−0·194	0·143	−0·180	0·042	0·069	0·120
Activity	0·319	−0·049	−0·282	−0·121	0·277	−0·144
Social behaviour	0·047	0·185	−0·042	−0·043	0·048	−0·195

TABLE III

Difference in Average Deviation

(a) *Aggression*

Source of Variation	Sum of Squares		Degrees of Freedom	Mean Square	Variance Ratio	Significance
Patients	177·096:		17:	10·417	30·60	p<0·001
Between orders of treatment		34·697	5	6·939	0·58	
Within orders of treatment		142·399	12	11·867		
Between periods ..	1·679:		5:	0·336	0·99	—
Linear regression ..		1·560	1	1·560	4·58	0·025<p<0·05
Higher terms ..		0·119	4	0·030	0·09	—
Treatments:						
Direct effects ..	*0·453*	0·352	5	0·091	0·27	—
Effect of preceding treatment	1·456	*1·557*	5	0·311	0·91	—
Error	25·529		75	0·340	—	—

(b) *Activity*

Source of Variation	Sum of Squares		Degrees of Freedom	Mean Square	Variance Ratio	Significance
Patients	211·841:		17:	12·461	45·43	p<0·001
Between orders of treatment		43·710	5	8·742	0·62	—
Within orders of treatment		168·131	12	14·011		
Between periods ..	3·292:		5:	0·658	2·95	0·01<p<0·025
Linear regression ..		1·519	1	1·519	5·54	0·01<p<0·025
Higher terms ..		1·773	4	0·443	1·62	—
Treatments:						
Direct effect ..	*3·133*	2·020	5	0·627	2·28	—
Effect of preceding treatment	3·031	*4·144*	5	0·829	3·02	0·01 p 0·025
Error	20·574		75	0·274	—	—

TABLE IV

Difference in Average Behaviour for Activity Only

Source of Variation	Sum of Squares		Degrees of Freedom	Mean Square	Variance Ratio	Significance
Patients	258·269:		17:	15·192	15·51	p<0·001
Between orders of treatment		83·504	5	16·701	1·15	—
Within orders of treatment		174·764	12	14·564		
Between periods ..	6·949:		5:	1·390	1·42	—
Linear regression ..		0·007	1	0·007	0·01	—
Higher terms ..		6·941	4	1·735	1·77	—
Treatments:						
Direct effects ..	*16·379*	13·607	5	3·276	3·34	0·001<p<0·01
Effects of preceding treatment	8·187	*10·958*	5	2·192	2·24	—
Error	73·475		75	0·980	—	—

be noted that the average for the preliminary period (no drug) is for 39 observations, whereas the averages for the drugs are derived from 21 observations. The preliminary period also differs in occurring for all patients at the same time, and during a fine weather period. It has previously been noted (Craft, 1957a, page 856) that rating scores mount when hyperactive patients are confined indoors during bad weather.

Table III gives the Analysis of Variance for the Latin square for deviation, and Table IV for behaviour respectively. These analyses represent a comparison of the six drugs and do not include any information from the preliminary period.

DISCUSSION OF RESULTS

(a) *Aggression*

From Table III it may be seen that the mean square due to variation between patients compared with the error mean square gives a value for the variance ratio of 30·60. This corresponds to $p+0 \cdot 001$ from the F-tables, indicating a highly significant difference between patients. Breaking this down into variation between orders of treatment (that is, within the groups of six patients) and within orders of treatment between groups of six shows that there is no evidence that the patients in different groups differed more than those in the same group, although in both cases there is still a significant difference when compared with the error mean square. That is merely saying that the 18 patients are not a particularly homogeneous group as far as their deviation from normality in aggression is concerned. However, because of the design of the experiment this lack of homogeneity, which might have masked any possible treatment effects, may be separated from the rest of the analysis. The fact that the variation between orders of treatment is comparable with that within orders of treatment suggests that there is no difference in rating between the nursing staff on the two villas.

Considering the variation between the six 3-week periods the mean square of 0·336 due to this cause corresponds to a variance ratio of 0·99, which would seem to indicate that there is no difference between the periods. If, however, we consider the possibility that the deviation in aggression may steadily increase (or decrease) over the 18 weeks under consideration we find that there is, in fact, a significant increase during this time indicating a steady increase in aggression ($F=4 \cdot 58$, $0 \cdot 025<p<0 \cdot 05$).

It is in considering the possibility of a steady increase that this analysis differs from that of Raymond. This extra analysis merely involves breaking down the sum of squares due to the difference in periods into five parts due to linear variation, quadratic variation and so on up to quintic variation, each with one degree of freedom. None of the terms higher than the linear were here significantly different from zero.

It may be noted here that it is possible that had Raymond *et al.* analysed their results in this way, they might have obtained a similar result. Their sum of squares due to variation between periods is large enough to provide a significant linear term, but it would need the original data to enable this sum of squares to be broken down in the appropriate manner.

Continuing the interpretation of Table III the remaining two variance ratios indicate no significant difference between treatments or between their residual effects. This is not to say that the drugs themselves have either no direct or no residual effect. This is almost certainly not the case, since otherwise a steady increase with time would be unlikely. The analysis merely says that the drugs appear to act in a similar way.

Comparing the average deviation for the whole period during which drugs were administered (2·007) with that for the preliminary period (1·591) there is a difference of 0·416. Because these preliminary observations are additional to the main design, it is not possible to eliminate the difference between patients as was done in the main analysis. These patient differences inflate the standard error of the averages to such an extent that a real difference of this order would be difficult to detect, and it is only possible here to say that although there appears to be no difference between the drugs themselves, there has been a steady increase in aggression over the whole 165 days of observation. This is possibly due to the cumulative effect of the drugs, also possibly due to external circumstances such as weather, a cumulative effect of confinement indoors, or even to a steady shift in the standards of judgment of the nursing staff.

(b) *Activity*

The analysis in Table III of the deviations in activity shows a pattern similar to that of the deviations in aggression, with one exception. As before there is, as might be expected a considerable difference between patients generally ($F=45·43$, p $0·<001$), but not between patients in the different groups of six. In this case, however, there is also a significant difference between periods, but since this is entirely due to a linear trend ($F-5·56$, $0·01<p<0·025$) the conclusion is similar to that for aggression. The linear trend is here negative, indicating a steady decrease in deviation from normality during the experimental period. Reference to Table IV shows that this effect disappears when the behaviour of the patients is considered indicating that this is a true tranquillizing effect.

Further analysis shows that the drugs show a significant difference between themselves with respect to residual effects (Table IIb). Application of the test for extreme values (see for example, Pearson and Hartley, 1954, p. 49–51 and p. 173) to the second row of Table IIb shows that the drugs may be divided up into two classes; amylobarbitone with promazine, and the others. Amylobarbitone and promazine have a greater *positive* residual effect than the remainder of the drugs. Since, however, the general effect of the drugs as indicated by the period difference seems to be in reducing activity, this is equivalent to saying that amylobarbitone and promazine appear to be *less* effective in their residual effect than the others used.

The analysis of the average behaviour of the patients in Table IV exhibits one notable difference from the analysis of average deviation. The significance of the residual effects is no longer apparent, although the value of 2·24 for the variance ratio is fairly close to the appropriate 5 per cent. significance level of 2·35. There is, however, a fairly significant treatment difference ($F=3·34$, $0·01<p<0·025$), which is largely due to the effect of prochlorperazine. Using the extreme value technique already referred to, the value of —0·780 found for prochlorperazine is significantly lower than the other treatment effects. It would appear, therefore, that this drug has a useful tranquillizing effect compared with the other five drugs. It may be noticed that it also has a strong negative residual effect.

There is some evidence that the average deviations for the periods during the administration of the drugs are different from the average deviation during the preliminary period, though an accurate comparison is not possible here. It may be concluded that there is some prima facie evidence that all the drugs have a similar small effect in decreasing activity with reservation for amylobarbitone and promazine noted above.

(c) *Social Behaviour*

The analysis does not reveal any significant differences between the drugs or periods, although there is, as might be expected, a difference between patients. Although a strict comparison with the preliminary period is not possible, it appears unlikely that any real difference between this period and the drug administration periods exists.

(d) *Toxic reactions*

Toxic reactions occurred with several drugs. Undue sedation with staggering gait was reported among six patients with promazine, acetyl promazine, promethazine, and prochlorperazine. Two unpleasant hypotensive reactions among patients on promazine hydrochloride necessitated a reduction of dose to a maximum of 300 mg. early in the trial. Two further patients had hypotensive episodes on acetyl promazine some hours after grand mal fits. Four instances of parkinsonism occurred in the eighteen patients on prochlorperazine, each recovering with alteration of dose, but suggesting that a dose of 150 mg. daily was near the upper limit for this group of patients. None of these toxic reactions occurred among the adolescents and none lasted more than two days.

CONCLUSIONS

A detailed statistical analysis shows that during the trial there was a steady increase in the deviation from the normal on the rating scale for aggression. This might possibly be due to the cumulative effect of the drugs, but more probably is due to continuing confinement in winter months. The activity rating scale showed a steady decrease in activity perhaps due to the cumulative effects of the drugs. Prochlorperazine appeared to have a significant effect in reducing activity where $0{\cdot}001 < p < 0{\cdot}01$, an absence of undue sedation, with a return towards normally acceptable behaviour. However, at the dose used, 150 mg. daily, there were four parkinsonism reactions among eighteen patients. A further analysis of the scale of activity suggested that there was some evidence that amphetamine, promethazine and acetyl promazine had a similar, small effect in decreasing activity. Since toxic reactions of varying degree occurred among four of the six drugs used, there is evidence to suggest that dosage with these drugs was at least adequate. Amphetamine, promethazine, acetyl promazine and prochlorperazine were found to have a definite residual effect after discontinuation of the drug, and this should be borne in mind in the planning and analysis of other trials. The Latin square type of trial seems to be relatively efficient in assessing a number of drugs over a short period of time with the same patients, also in enabling one to compare drugs with similar actions fairly stringently against each other. One considerable disadvantage of such trials is the need to balance the experiment; if one or two of the patients had been transferred or lost from this trial, much of the statistical analysis would have been impossible. It is probable that under the circumstances the significant effect of prochlorperazine would not have been uncovered.

SUMMARY

A Latin square trial is reported, assessing the effect of six tranquillizing agents among mental defectives, using three rating scales. Statistical analysis suggested that all agents used had some slight effect in decreasing the hyperactivity shown by these predominantly brain-injured patients during winter months. A significant ($0 \cdot 001 < p < 0 \cdot 01$) reduction on the activity scale was shown by prochlorperazine at 150 mg. daily, but at this dosage four parkinsonian syndromes resulted among eighteen patients. Within the limits of this study the drug appears to be the most effective tranquillizer and deserves further investigation.

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TESTING FOR INTELLECTUAL IMPAIRMENT—SOME COMMENTS ON THE TESTS AND THE TESTERS*

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INTRODUCTION

PSYCHOLOGISTS are frequently asked to say whether, in the case of a particular patient, there is evidence of organic dementia. At first sight this would not appear to be an unreasonable request. A considerable amount is known about the performance of normal people on tests of cognitive function and it might be supposed that people who work with tests of human ability should be able to modify their procedures to the purpose of diagnosing intellectual deterioration when this is not obvious on routine neurological or psychiatric examination. Moreover, it might seem that the frequent presence of independent criteria of brain damage provided by neurological and neurosurgical investigations should facilitate the development of competent diagnostic clinical psychology in the neurological field.

Nevertheless, the reaction to this practical challenge has in some psychological circles been pessimistic. Thus, in a Critique of Psychological Approaches to Brain Damage, Meyer (1957) writes: "It would appear that the clinical psychologist's contribution to the problems of the neurosurgeon and the psychiatrist is of little value owing to the lack of proper diagnostic tools."

This "lack of proper diagnostic tools" was extensively reviewed by Yates in a paper entitled "The Validity of some Psychological Tests of Brain Damage" (1954). He came to the conclusion, not only that no adequate test of brain damage existed, but also that none was likely to be devised as long as the approach was a purely empirical one.

And yet, intellectual impairment resulting from cerebral lesions is often gross and so obvious as to render psychological tests of deterioration superfluous. Moreover, there may be every degree of impairment, from obviously pathological dementia to defects which are so slight that they can be inferred only with hesitation. What then are the difficulties which prevent us from testing for what might be called sub-clinical dementia?

THE INADEQUACY OF CURRENT TESTS

The criticisms that have been made by Yates are representative of the position of a number of British clinical psychologists. Firstly, most standardized tests result in too many misclassifications to be regarded as adequate diagnostic instruments. This is an objection with which few people would quarrel. A test may discriminate between brain-damaged and other groups statistically at a high level of confidence but nevertheless not give a clear-cut answer in the individual case. Valid and reliable cutting scores are notoriously difficult to devise in this field and one would do well to enquire why this should be the case.

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* Based on a paper read at the Annual General Conference of the British Psychological Society in April 1958.

Part of the answer is supplied by another objection: there are very good reasons for supposing that the effects of cerebral lesions on intellectual function are not of a unitary nature. This is a point to which it will be necessary to return but it may be pointed out at once that any single test which purports to measure the effects of brain damage, irrespective of the site and nature of the lesion, assumes a unitary theory of cerebral dysfunction.

Another objection is that, in the validation of these tests, important variables such as age, intelligence and sex have not been controlled. One may add to the list of uncontrolled variables by mentioning such things as concurrent emotional disturbance (which may be of various kinds), and motivational anomalies which may affect test performance.

A fourth criticism, one which is most popularly levelled at the Rorschach test and the Goldstein-Scheerer tests, is that some tests allow of subjective methods of scoring.

Fifthly, Yates suggests that no test of brain damage is likely to be satisfactory unless it is based on an adequate theory of brain function.

The aim of test designers who are aware of these difficulties would appear to be to devise a test of brain damage which is administered, scored and interpreted in a standardized manner, which takes into account all relevant variables, which is based on an acceptable theory of cerebral function and which, when rigorously applied, results in a minimal number of misclassifications. Such an aim would be admirable if it were realistic.

It is important to bear in mind that the problem is a practical not a theoretical one and that the requirements of diagnostic practice are not identical with those of fundamental research. Thus, for research purposes, we may suspend judgment as to how the brain works for a very long time indeed. It is not however appropriate to suspend judgment indefinitely on diagnostic problems, solely on theoretical grounds.

It is suggested therefore that the conception of tests of brain damage outlined above is an inappropriate one. In particular it is submitted that, for practical purposes, some kinds of evidence are better assessed by a human brain than by a standardized psychological test. In examining for brain damage, is it appropriate for the clinical psychologist to identify himself with his tests? That is to say, should the tests be regarded as, if not fool-proof, then at least psychologist-proof?

There are indeed those who, aware of the limitations of tests applied to abnormal people, suggest that the clinical psychologist should limit himself to purely experimental work either in fundamental research or else in protracted studies of individual cases. This point of view was expressed by R. W. Payne (1957). In accordance with the approach recommended by Shapiro in 1951, he advocated the application of controlled scientific method to the study of symptoms exhibited by individual patients. Symptoms should be carefully defined, hypotheses put forward to explain them, deductions drawn from the hypotheses and experiments carried out to verify or refute the hypotheses. From the practical point of view, two main criticisms of this approach may be made. Firstly, as is generally admitted, such a procedure is extremely time consuming; secondly, the method makes no provision for eliciting symptoms as opposed to analysing symptoms which are known to be present. However, Payne expressed the view that this is the only thing we can usefully do.

Implications of Administering a Standardized Test of Intellectual Impairment

The present writer believes that there is an alternative approach and this may be introduced by first considering some of the implications of administering standardized tests to brain-injured patients.

The greatest difficulty is probably that, in testing brain-injured patients, one is dealing with an extremely heterogeneous population. It is well known that if a test is given to a random group of brain-injured patients the test results are skewed, some performances being indistinguishable from the normal, others being more or less abnormal. It is of course possible that some unitary and measurable intellectual change is involved in all cases of brain damage but it can be said with confidence that, whether or not this is the case, the existence of different syndromes of disability is much more obvious. This is particularly apparent if one considers extreme cases such as obvious dysphasias, apraxias, agnosias or organic amnesic states. These and other syndromes vary, however, not only in their degree, but also in the constellation of symptoms present in any one patient. Although it may be possible to describe a comparatively small number of typical syndromes of intellectual disability associated with brain damage, any individual patient may show, not only mixtures or fragments of these syndromes, but also anomalous disabilities resulting from an interaction between them. It is very difficult to see how a practicable test battery could be devised which accurately identified the presence and nature of disability resulting from a cerebral lesion, and at the same time met the requirement of the more extreme psychometricians that the diagnosis should be rigidly proscribed by the test scores.

These considerations alone cause one to wonder whether such a test battery is a practical proposition. But in addition one has to take into account other factors which would distort test results. One of these is the emotional disturbance which frequently accompanies cerebral damage. How is one to deal with the catastrophic reaction to failure described by Goldstein (1942)? And what of the impulsive unselfcritical performance that is sometimes observed in cases of frontal lobe lesions? Similarly, in many organic patients, fatigue develops rapidly and misleadingly after a comparatively short spell of testing. Others are distractible if the rules of test administration are followed rigidly, although their attention may be held if the procedure is modified in a manner appropriate to the individual case.

One of the most important disadvantages of any standardized test of brain damage is that much information tends to be missed that otherwise might be utilized. The type of information most likely to be missed is that which is frequently referred to as the "qualitative" aspects of test performance. This common-sense distinction between qualitative and quantitative is of course artificial in the present context and the distinction may be reduced to a difference in the ease of quantifying the different types of response to a test.

Firstly, the nature of the failure which results in a low score is usually not apparent from the score alone. Thus, on a test of rote verbal learning, one patient may be impaired as a result of a slight but strongly perseverative error whereas errors produced by another patient obtaining an identical score may be extremely variable. The distinction is not trivial since the latter is more typical of dysphasia and is thus of localizing value. Similarly, failure on the Kohs blocks type of test may be due either to failure in principle or to an inability to cope with a particular detail. If a mild constructional apraxia is to be recognized, it is essential that differences of this type should not pass unnoticed by the tester. Again, if on this type of test the trial is discontinued as soon as the conventional time limit is exceeded, the opportunity to distinguish between depressive and organic intellectual impairment may well be lost.

Secondly, in the usual standardized test situation it is often not possible to make allowance for irrelevant causes of failure. Poor co-operation is an obvious example of this and, in fact, there are probably few psychologists who do not attempt to assess co-operation and utilize this assessment in interpreting the test results. Nevertheless, it is worth pointing out that the procedure for such an assessment is never standardized, and could not readily be standardized. It seems right to enquire whether, in the case of testing for dementia, there are not other equally important variables which cannot readily be standardized but which must be allowed for if a valid assessment is to be made.

Inadequate comprehension of standardized instructions is by no means uncommon in psychiatric and neurological patients and may result in artifactually low scores on the test involved. A constant effect of instructions, in terms of "task set", is clearly more important than constant wording of instructions, and indeed the former may be regarded as the aim of the latter. In a population as heterogeneous as that under consideration, constant wording is often insufficient to ensure constant effect of instructions.

A third defect of the standardized psychological test for brain damage is that anomalous responses, produced by the patient but not specifically tested for, are likely to be ignored. An amnesia for words as names may sometimes emerge on a picture completion test athough not on the type of naming test usually employed by the neurologist or psychiatrist. Similarly, diagnostically important inconsistencies in performance are frequently not reflected in test scores. Thus the interpretation placed on the ability to repeat 7 digits forwards and 5 backwards should be quite different from that placed on the ability to repeat only 5 digits forwards but 7 backwards. This particular inconsistency could readily be allowed for in the standardization but it must be emphasized that the number of possible inconsistencies on a fairly small battery of tests such as the Wechsler Bellevue Scale is exceedingly high. These can be recognized by an experienced examiner when they occur but cannot readily be catered for in advance in the standardization. Nor will the same interpretation be placed on all inconsistencies. Thus the example already cited may reflect emotional interference; but inconsistency in the sense of success when tested verbally but failure when tested in the visual modality, may point to a perceptual disability of localizing value.

Fourthly, a practicable standardized test can allow very little latitude for the examiner to ask supplementary questions which are dictated by the patient's previous response. Such a procedure may however be extremely valuable when testing people who are organically or emotionally impaired. Thus the approximate and unreflective answers which may be given by a euphoric patient on a vocabulary test can result in a score which grossly underestimates the patient's ability. Carefully chosen supplementary questions, however, may do much to reveal ability which would otherwise be masked by the effect of the test taking attitude. Supplementary questions and special promptings aimed at testing the limits of a particular disability are frequently called for when anomalous responses are produced by neurological or psychiatric patients and these supplementary procedures tend to be dictated by the previous response of the testee.

We are faced therefore with the following difficulty. The effects of brain damage on intellectual performance are various and are often complicated by and difficult to disentangle from the various effects of emotional disturbance. Nevertheless, particular syndromes of disability, when present in an extreme form, can be identified without the need for psychological tests. How should psychological tests be constructed and used in order to make possible the identification of organic intellectual deficit when this is not obvious to the psychiatrist or neurologist?

The Gap between Theory and Practice

Now it might *in theory* be possible to devise a battery of tests standardized as to administration, scoring and interpretation, which would accommodate the complexity of test procedure necessary to identify most cases of organic intellectual impairment. It must be stressed, however, that in most cases of mild intellectual impairment the logical structure of any thorough assessment of the evidence is extremely complex, and if formalized would result in a test battery incorporating a formidable decision procedure for administering successive tests. This decision procedure would be of prohibitive complexity and, in addition, there would be a need for an extremely complex system of weightings if the various possible combinations of response patterns are to be recognized and assessed within the framework of the scoring system. All this might, in theory, be done but only in the same sense that it is possible to formulate a decision procedure for playing the perfect game of chess.

The theory of finite games is of some relevance here. In theory, both noughts and crosses and chess are amenable to methods of analysis which would yield an infallible decision procedure for play. In the case of noughts and crosses this is not only possible in theory but is a practicable proposition, and human skill at noughts and crosses then becomes unnecessary. In the case of chess, although the appropriate methods of analysis are available, the total number of possible outcomes associated with each of the large number of possible moves for any one of the extremely numerous possible dispositions of pieces on the board, prohibits the formulation of an invincible decision procedure as a *practicable* enterprise. In practice, chess players treat chess as an infinite game, and experienced and skilled players can defeat opponents guided *solely* by any available formal rules of procedure. The reason for this is fairly obvious but is worth stating if the relevance of this example to testing for deterioration is to be made clear. The reason is that the human brain is the best available instrument for assessing some types of evidence.

Although a completely standardized procedure is less fallible than human judgment where all relevant parameters can be accommodated in the standardization, the reverse is often the case when the problem exceeds a certain level of complexity. At this level, it seems essential to relegate a certain proportion of the responsibility for assessing evidence to a human agent. Since the validity of standardized tests of intellectual deterioration is generally acknowledged to be low, this proportion should be considerable in the case of the psychological test procedure under discussion. Human error must be tolerated if other sources of error are greater.

A Comparison with Medical Diagnosis

At this point it is useful to compare the problems of diagnostic clinical psychology with those of clinical medicine. The diagnostic problems encountered in medicine are comparable in complexity with those encountered by the psychologist who attempts to assess intellectual impairment. It is by no means unusual in medicine to encounter diagnostic problems where there is no single objective test available which will give a yes or no answer as to the presence of a particular disease. Often the interpretation of "qualitative" evidence is essential, as in the diagnosis of early Parkinsonism and as in the interpretation of X-ray photographs. Often also it is necessary to assess various lines of evidence in arriving at a diagnosis, as in diagnosing some atypical cases of appendicitis.

If only fully standardized tests should be trusted in clinical psychology, this should apply *a fortiori* in medicine since bad doctors are clearly more dangerous than bad psychologists. And yet, in medicine "clinical judgment" is highly esteemed. Having regard to the complexity of the problem, it is the writer's view that this state of affairs is appropriate. It would seem also to be appropriate that clinical judgment, in the sense of the rational assessment of unique evidence, should be valued in testing for intellectual impairment, since the manifestations of cerebral dysfunction are multiform and subject to considerable influence from uncontrollable variables such as emotional disturbance.

Among some of the more articulate British clinical psychologists, however, there is an unreasonable tendency to regard the exercise of human judgment in diagnostic work as something to be avoided even when it is useful. Writing of what he describes as the "art of psychology" (and by this he means any diagnostic psychological procedure which is not either a fully standardized test or a scientifically watertight experiment) Payne states: "This method of work also depends on the capacity of the individual doing it and there would seem to be no way of establishing this without conducting an elaborate investigation into the validity of each individual psychologist as an oracle."

If this principle were applied to general medicine, many diagnoses which should be and are made, never could be made. Clinical psychologists are able to insist upon being indecisive only because other people are available who are more willing to take decisions and less able to avoid taking decisions. Nevertheless, careful but flexible psychological testing by an examiner with extensive experience of psychiatric and neurological patients fairly certainly provides the most trustworthy opinion available as to the degree and nature of intellectual impairment in a particular patient. The fact that the examiner who is best equipped to do this has not been validated as an oracle is no adequate reason for his declining the task especially if, failing him, an even more Delphic pronouncement on the case will be made by somebody else.

The Identification of Syndromes

A more satisfactory orientation towards this problem could perhaps be achieved if clinical psychologists took as their reference points not, as is usually the case, the normal performance on particular tests but the performance on these tests of patients who exhibit easily recognizable syndromes of intellectual disability. Schooled in the theory of normal individual differences, many psychologists tend to use significant departure from the norm as the essential criterion for recognizing intellectual deterioration, and implicitly assume that the same test measures the same thing in all types of abnormal people, including those with grossly and variously modified nervous systems.

This could be defended if there were only one possible type of abnormality which could produce an inferior score on any given test, but this is indisputably not the case.

An alternative procedure is to examine test results with respect to their degree of approximation to known syndromes of intellectual disability. This procedure will assume that the tester is familiar with these easily recognizable syndromes in the test situation and that, when he is confronted with more difficult cases, he will be more concerned with identifying the behaviour he elicits on the tests with one or more of these syndromes than with recognizing the behaviour as deviating from the norm.

Clearly, much remains to be done in identifying the less obvious syndromes of intellectual disability and it must also be admitted that the method of extreme cases which is advocated here is bound to have its limitations. However, that which is known about gross and typical syndromes of disability should not be neglected in diagnostic work simply because no general theory of brain function is available (Yates, 1954) or because there is no test available which does not call for judgment on the part of the tester as well as for the ability to say whether a particular score is above or below a cutting score (Payne, 1957).

In conclusion, it should be said that the position outlined above refers solely to diagnostic work and is not intended to detract in any way from the necessity for the most careful application of scientific method in research in this field. It is however suggested that diagnostic clinical work and research in clinical psychology have different aims and call for different procedures.

SUMMARY

The assessment of organic impairment of intellect is a task which might be expected to be within the competence of a clinical psychologist. Nevertheless, recent literature on the subject contains statements by psychologists disclaiming their ability to do this with an adequate degree of validity. The difficulty appears to resolve into the problem of finding a foolproof standardized test. It is suggested here that these disclaimers are based on an inappropriate rationalization of the problem and that the assessment of intellectual deterioration calls, not for a single valid standardized test, but rather for a flexible test procedure and awareness of the relevant syndromes. This view is justified by a consideration of the multiform effects on intellectual performance of focal and diffuse cerebral lesions and also by a consideration of the focal disturbances of intellectual function already recognized by neurologists and utilized in diagnosis.

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PROLONGED APNOEA FOLLOWING E.C.T. MODIFIED BY SUXETHONIUM (BREVIDIL "E")

By

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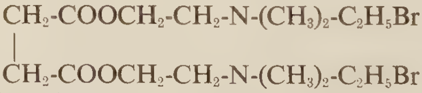
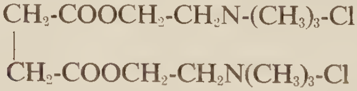
THERE have been numerous reports in recent years of prolonged apnoea resulting from the use of suxamethonium (succinylcholine, "Scoline") in general anaesthesia and for electroplexy. For example, Grant (16) described a case of respiratory paralysis lasting 2 to 3 post-operative days following an intravenous dose of 80 mg. of suxamethonium, and Cowan (7) reported a patient who underwent a partial gastrectomy and exhibited complete apnoea for 9 hours duration after only 25 mg. of suxamethonium had been given, and who subsequently died. Hurley and Monro (22) reported apnoea lasting for 10 minutes following 20 mg. of suxamethonium used as a relaxant for electroplexy, and for 20 minutes after decamethonium iodide.

Respiratory paralysis of a duration comparable to that recorded after suxamethonium, does not so far appear to have been described in association with suxethonium (Brevidil "E"), the di-ethyl derivative of suxamethonium, which is surprising in view of the close structural similarity between these two relaxants as indicated by their chemical formulae.



$$\begin{array}{l}CH_2\text{-}COOCH_2\text{-}CH_2\text{-}N\text{-}(CH_3)_2\text{-}C_2H_5Br\\ |\\ CH_2\text{-}COOCH_2\text{-}CH_2\text{-}N\text{-}(CH_3)_2\text{-}C_2H_5Br\end{array}$$

Suxethonium Bromide (Brevidil "E")



$$\begin{array}{l}CH_2\text{-}COOCH_2\text{-}CH_2N\text{-}(CH_3)_3\text{-}Cl\\ |\\ CH_2\text{-}COOCH_2\text{-}CH_2N(CH_3)_3\text{-}Cl\end{array}$$

Suxamethonium (Succinylcholine, "Scoline").

It is probable, however, that this complication is not unfamiliar to those psychiatrists who regularly use suxethonium for E.C.T. modification.

Pharmacologically, both relaxants act identically causing depolarization of muscle membranes at the myoneural junctions, a process normally reversed by serum pseudocholinesterases. Enzymatic hydrolysis of suxamethonium and suxethonium is effected by these cholinesterases, more rapidly in the case of suxethonium than suxamethonium (13), which makes the former an ideal muscle relaxant for use with electroplexy (4), (29), (11).

In spite of their advantages over other muscle relaxants used for electroplexy, short periods of apnoea following injections of Brevidil compounds have been observed by various workers. Blayney (4) reported apnoea lasting for 10 minutes following 30 mg. Brevidil "M" in a patient who was considered a poor risk, and Gillie and McNeill (15) referred to 3½ minutes apnoea after 100 mg. Brevidil "E". Fisher and Bannister (11) reported 5 minutes apnoea and Wolfers (41) 4 minutes apnoea after 90 mg. Brevidil "E", and the manufacturers (42) have referred to occasional instances of prolonged relaxation lasting beyond the usual 2 to 4 minutes.

Since there are no reports of prolonged apnoea following the use of the suxethonium brand of relaxants, the following cases are of interest, and indicate that suxethonium, like suxamethonium, is a powerful respiratory depressant, which may in certain circumstances produce not only apnoea but a profound degree of collapse.

Such a situation, occurring "out of the blue" during the routine performance of electroplexy, is an alarming experience for the doctor concerned, and sharply reminds us of the views of Sargant and Slater (34), who asserted that "to add pentothal and a relaxant to E.C.T. is to add two potential sources of danger each carrying its own risks of death".

Case 1

A male patient, F.A.W., was admitted in 1949, aged 53, suffering from a paranoid psychosis. A builder's labourer, he was in good physical health apart from a well-compensated mitral stenosis. His blood pressure was 170/100, but there were no other abnormal physical findings.

He had many fantastic grandiose delusions and ideas of reference which were mainly expressed towards the staff of the hospital. From 1949-51 he underwent several courses of "straight" electroplexy, resulting in marked temporary mental improvement, though he soon relapsed. No untoward effects were noticed after any of these treatments. In 1954 mass radiography revealed bilateral tuberculous infiltration of both apices and he received a course of chemotherapy. Throughout this time his general health remained excellent. In October 1957 he became increasingly boastful and paranoid, and imagined that his name and personal affairs were being broadcast over the radio.

Chlorpromazine 100 mg. t.d.s. was commenced with slight mental improvement, but this was omitted on the morning of treatment. On 1 November, 1957, he was given E.C.T. modified, in view of his chest condition. He had a well-relaxed fit, using the Ectonus technique, following 75 mg. Brevidil "E" given intravenously. No anaesthetic was used. Following this, his condition gave rise to increasing alarm and he became collapsed and pulseless, his blood pressure falling to 100/74. A fatal outcome was expected for he did not breathe spontaneously for approximately one hour, and when breathing recommenced it was initially shallow and jerky. He also developed bouts of auricular fibrillation.

Artificial respiration was performed manually, and with an oxygen and 5 per cent. CO_2 mixture under positive pressure using the Lucas breathing apparatus. Repeated intravenous injections of nikethamide were given (2 ml. in 10 ml. of saline) with little apparent effect, possibly because the dose used was too small. Unsuccessful attempts were also made to induce respiration by means of sub-shocks using the Ectonus machine, and to raise the blood pressure by intravenous injections of methedrine. Consciousness returned after one hour and 15 minutes, but apart from a slight headache the patient noticed no after effects. Blood taken for pseudocholinesterase estimation during the period of apnoea gave a value of 27 units. Following this single electric treatment the patient's mental condition improved markedly and he was able to return to employment in the hospital. A further pseudocholinesterase estimation on 27 June 1958 showed a value of 43 units. Liver function tests were regarded as normal, though the alkaline phosphatase was slightly raised (14 K units).

On 29 June the patient died suddenly while attending a church service. Post-mortem indicated that the cause of death was a small posterior myocardial infarct. There was no evidence of previous myocardial damage or mitral stenosis, though some thickening of the mitral valve was noted. The biapical tuberculous lesions were of minimal extent, and the liver appeared completely normal.

Case 2

A male patient, C.H.M., was admitted in August, 1955, aged 41, suffering from a schizophrenic psychosis. Physical examination on admission was normal and he appeared to be in excellent health. For the preceding eighteen months he had been developing ideas of reference and loss of concentration, and had noticed lack of manual skill together with troublesome aural hallucinations which gave a running commentary on his actions. He was obsessional and excessively preoccupied with his bodily functions.

Between April and November 1956 he received treatment with a variety of tranquillizing preparations (chlorpromazine, reserpine, suavitil, ritalin and meprobamate), but these had all been omitted by 26 November, when there appeared to be marked mental deterioration.

On 28 January, 1957, he received E.C.T. well modified with 75 mg. Brevidil "E". No anaesthetic was used, and no other drugs had been given except atropine gr. 1/100 ¾-hour beforehand. Following this treatment he did not breathe spontaneously for 45 minutes, and his pulse became irregular and varied in rate between 60 to 120 per minute. Artificial respiration was conducted manually throughout this period.

Afterwards he complained of no untoward effects apart from slight weakness and loss of sensation in the right leg. He was regarded as a case of hypersensitivity to Brevidil "E", and a number of pseudocholinesterase levels were performed between 28 January and 4 February which varied between 13·2 and 21·2 units. Liver function tests were carried out showing, total protein 6·7 G. per cent., albumin 4·7 G. per cent., globulin 2·0 G. per cent., albumin-globulin ratio 2·3/1, alkaline phosphatase 3 K units, urea 28 mg. per cent., total cholesterol 161 mg. per cent., free 53 mg. per cent., ester 108 mg. per cent., free-ester ratio 1/2·0, the low phosphatase level indicating a very slight degree of liver damage.

On 13 May the patient received a further E.C.T., this time unmodified in view of the previous experience, following which there were no respiratory complications. This caused marked mental improvement, and he has remained fairly well up to the time of his recent discharge.

Case 3

A male patient, R.W.J., aged 35, was admitted on 18 April, 1958, suffering from a schizophrenic psychosis.

Physical examination on admission was normal and he appeared to be in excellent health. There was no history of jaundice, and he had not at any time been exposed to chemical agents such as insecticides which are known to lower the pseudocholinesterases. Treatment with chlorpromazine 50 mg. t.d.s. was commenced on 19 April, but was omitted on treatment mornings.

On 21 April, he was given E.C.T. by the Ectonus technique using 50 mg. Brevidil "E". Atropine gr. 1/60 had been given 45 minutes previously, together with sodium amytal gr. 6 as he was apprehensive. The convulsion was well relaxed, but he became extremely cyanosed, and later white and shocked in appearance, and his pulse practically disappeared at the wrist. Altogether some 10 minutes elapsed before spontaneous respiration returned, which was initially shallow. During the apnoea artificial respiration was continued manually, assisted by oxygen given under positive pressure using the Lucas apparatus. The patient complained of no after-effects.

Because of this incident a further 8 treatments were given unmodified without any respiratory difficulties. After this he became increasingly hypochondriacal, and imagined his thoughts were under the direct control of wireless and television. He demanded to leave the hospital, and asserted that he would not consent to further E.C.T. unless he was given injections prior to treatment.

On 10 June E.C.T. was again given using 75 mg. Brevidil "E" following which he did not breathe for 4½ minutes. Blood taken during the apnoea contained 26 units of pseudocholinesterase. Three days later he received a further treatment using 50 mg. Brevidil "E" and this time the apnoea lasted 7½ minutes. The pseudocholinesterase was now 24 units. Liver function tests were reported as showing normal results.

He noticed no after-effects following these last two treatments, but insisted on handing in his notice of departure. Investigation of this case had therefore to be discontinued.

Discussion

It will be observed that all three patients had low serum pseudocholinesterase levels at the time and shortly after the respiratory difficulties were encountered, and that recovery was uneventful when unmodified E.C.T. was given. It is generally assumed that the distress (apnoea, coma etc.) is caused by the accumulation of acetylcholine in the body, because of cholinesterase inactivation produced by the muscle relaxant (suxethonium or suxamethonium), and that the removal of the drug by the pseudo-cholinesterase normally curtails its effect.

Mean normal values for pseudocholinesterase of adult serum have been given at 76·7 units (25), and range between 53 and 102 units (39), and at this laboratory anything over 45 units is regarded as a normal value. (Units refer to microlitres of CO_2 evolved per minute per ml. of serum or whole oxalated blood, at 30° C.) The pseudocholinesterase is estimated by means of the Warburg Manometric Technique, as described by Aldridge and Davies (1). This is a micro-method which requires careful handling and considerable experience, but the same operator can achieve a high degree of reliability. Values between 50 to 80 per cent. of the normal figure for a particular patient are usually symptomless, but mild symptoms are likely to occur when the value falls between 20 and 30 per cent. of normal, and severe symptoms when it lies between 5 to 10 per cent. of normal. Generally values below 30 units would be expected in prolonged comata, but it is emphasized that the serum pseudocholinesterase level is characteristic for each person. A drop therefore from 80 to 50 units may be just as significant as a fall from 50 to 30 units. The body, as in other biochemical balances, gets attuned to a certain level of acetylcholine, and shows symptoms when this level is appreciably altered, analogous to some diabetics who go into hypoglycaemic coma at blood sugar levels around 250 mg. per cent. Once inactivated, the pseudocholinesterase regenerates itself slowly, and may take a month or more to reach its previous level. In Case I, therefore, 43 units is likely to be the normal pseudocholinesterase level, taken six months following the prolonged coma.

It has been stated (2), that in the same individual a fixed dose of suxamethonium given at suitable intervals, produces a constant response, and in any given person the duration of the paralysis increases with increasing doses. (38), (9). With suxethonium, however, Blayney (4) noted no great difference in the duration of activity when the dose employed was just sufficient to control the convulsion, or large enough to produce complete paralysis, and it was therefore his practice to have his patients completely relaxed, using doses ranging from 55 to 140 mg. of the active cation of Brevidil "E". By contrast Hurley and Monro (22) noticed no symptoms when the dose was reduced in a patient who had previously shown "scoline apnoea", and did not therefore regard this complication as a deterrent to future modified E.C.T.

Evans *et al.* (10) and Bourne *et al.* (5) demonstrated an inverse relationship between the duration of apnoea following suxamethonium and the serum pseudocholinesterase, but this finding has not been universally confirmed, for Argent *et al.* (2) observed that the pseudocholinesterase level in six out of seven patients who exhibited prolonged apnoea after suxamethonium was not depressed to such an extent that the reduced enzyme level could be held solely responsible. Conversely Foldes *et al.* (13) noticed no cases of excessively prolonged apnoea following suxamethonium in a group of patients showing reduced cholinesterase activity due to liver disease.

Wolfers (40) reported a case of suxamethonium sensitivity in a patient with a normal pseudocholinesterase level, and imagined that susceptibility to the drug could vary in the same patient from day to day, suggesting that the subject was more complex, and that the duration of apnoea must depend upon factors other than pseudocholinesterases. These have been listed by Foldes and Rendell-Baker (12) and include:

1. Accumulation of succinyl monocholine. This results from the hydrolysis of succinylcholine (succinyl-dicholine) by the plasma pseudocholinesterases.
2. Central respiratory depression by succinylcholine.

3. Central respiratory depression by general anaesthetic agents.

4. The production of succinylcholine "receptor block" rather than depolarization at the motor end plates. This would explain the beneficial results that sometimes attend the use of neostigmine in these cases of apnoea. (32), (33), (20).

5. Failure of redistribution of succinylcholine from the neuromuscular end-plates.

6. Persistent changes after removal of succinylcholine from the end-plates (refractory muscle).

7. Controlled respiration resulting in acapnia and exhaustion of the Hering-Breuer reflex.

The level of serum calcium which Irwin *et al.* (24) found to be lowered in animals with succinylcholine apnoea, may play a role which might profitably justify further investigation. The alteration of the patient's pH to the acid side resulting from acetylcholine accumulation leading to a slower rate of destruction of the drug (18), is likely to be an important factor in these conditions.

In these cases it seems probable that the serum pseudocholinesterase played an important part, since it was found to be greatly reduced in all three. In none was an anaesthetic (thiopentone) used, though in Cases 1 and 3 tranquillizers had been given up to the day before treatment, but in Case 2 they had been omitted for two months preceding E.C.T. It is known, for instance, that chlorpromazine and reserpine increase the risk of fatalities in patients undergoing E.C.T. and hence administration of these drugs should be suspended for a few days before commencing treatment (26). However, estimates of chlorpromazine urinary excretion indicate that a week or more may elapse before excretion stops after withdrawal of the drug. This means that tissue saturation with this drug may last for a considerable period, a point worth noting should there be any untoward effects in patients receiving muscle relaxants who have recently been treated with chlorpromazine.

It is also possible that in Cases 1 and 3, hyperventilation may have prolonged the apnoea by causing acapnia, though in Case 1 this was minimized by using an oxygen and 5 per cent. CO_2 mixture. The dangers of prolonging the apnoea brought about by relaxants by enthusiastic over-ventilation have been stressed by Gillie and McNeill (15), though in Case 2 this was minimized by using manual artificial respiration without oxygen.

It is emphasized that all three patients were apparently in good physical health when they received electroplexy. Low serum pseudocholinesterase levels have frequently been reported in patients described as "clinically ill", and Hodges (20) has suggested that pseudocholinesterase appears to be a critical index of the patient's physical state. Other causes of low serum pseudocholinesterase levels (amongst which are liver disease and insecticide poisoning) have been mentioned by Impastato and Berg (23) who also referred to low levels in 1 per cent. of normal subjects. Both Cases 1 and 3 had no history of liver disease and gave normal liver function test results, though in Case 2 mild liver disturbance was suspected, though there was no history or clinical evidence to corroborate this.

Forbat *et al.* (14) showed that low serum pseudocholinesterase levels might have a familial incidence, but it has not been found possible to investigate these patients from this aspect.

We wish to emphasize that to encounter a patient who exhibits prolonged apnoea following the use of these muscle relaxants (suxamethonium, suxethonium) during the routine performance of electroplexy, is an alarming experience, especially when the operator is working single-handed as so often is the case. A recent questionnaire (43) (results to be published elsewhere) circulated to over 50 mental and teaching hospitals, showed that at 60 per cent., E.C.T. was given modified with pentothal and scoline, and that relaxants were used at 85 per cent. Suxethonium was used at some 22 per cent. of hospitals. In spite of this, at nearly 50 per cent. of the mental hospitals we approached, the services of a Consultant Anaesthetist were not available during the routine performance of modified electroplexy. This we feel must tax considerably the experience and resources of the Psychiatric Registrar, who is surprisedly called upon to resuscitate one of these near moribund cases exhibiting hypersensitivity to muscle relaxants, especially in the absence of a specific antidote.

Some points concerning prevention and treatment

The treatment of prolonged apnoea following suxethonium (and other depolarizing muscle relaxants) is complicated, for as yet no specific antidote exists (29), (4), but the following points may be of assistance to the doctor called upon to cope with this emergency.

1. The maintenance of adequate respiration. This is of prime importance. Measures should be taken to ensure that there is an efficient airway, and artificial respiration should be continued manually and with an oxygen and 5 per cent. CO_2 mixture under positive pressure, until spontaneous respiration occurs.

2. The use of a test dose of muscle relaxant given as a routine to all cases before modified electroplexy, to ascertain whether any undue sensitivity occurs. This had been described in the case of suxamethonium by Impastato and Berg (23), who suggested giving a test dose of 5 mg. scoline to all patients, and from the response calculating the optimum dose for muscle relaxation. However, in view of the findings in a patient reported by Calvert *et al.* (6), who only exhibited prolonged apnoea following a dose of suxamethonium in excess of 40 mg., and who showed no response to a test dose of 10 mg., it is doubtful whether this time-consuming procedure would be justifiable as a routine.

3. The transfusion of whole blood or serum to replace the pseudocholinesterase. This has been reported by Harrison *et al.* (19), and on other occasions, and Cowan (7) described a case who showed a dramatic response to fresh blood after failure of older blood. Pseudocholinesterase preparations ("Cholase") which artificially elevate the serum pseudocholinesterase, have been used with success. (6).

4. There have been a number of conflicting reports about the value of neostigmine in these cases. Blayney (4) maintained that this drug, being an anticholinesterase, would inhibit the *in vivo* destruction of suxethonium by cholinesterase and prolong its action, a view also shared by a number of writers in connection with suxamethonium and suxethonium (31), (37), (35), (18), (5), (10), (17), (28), (20), (36). However, Ruddell (32) claimed success with 6·0 mg. neostigmine in a patient who developed apnoea following succinylcholine and decamethonium, and recently reported (33) a further case in which there was an immediate return of respiration after 2·5 mg. of neostigmine had been given. Hodges (21) has drawn attention to raised end-plate resistance to depolarization by suxamethonium, and showed that it was independent of cholinesterase

activity. Such "depolarization block" has been encountered in children, in cases of myasthenia gravis, carcinomatous neuropathy (8), and electrolyte imbalance, and may be induced by various drugs (particularly in those with an inherent tendency). E.C.T. with muscle relaxants should be avoided wherever possible in these subjects, though anticholinesterase compounds (neostigmine) may be helpful, and we believe should be given to all cases showing prolonged apnoea.

5. Opinions also differ concerning the value of nikethamide in the treatment of these patients. Harper (18) and Grant (16) used nikethamide without effect, but Barron (3) was enthusiastic about the use of this drug, if given in doses of more than 10 ml. intravenously, frequently finding that more than 20 ml. were required, and referred to it as "a simple and effective remedy". We would emphasize that no apparent success attended the use of nikethamide in Case 1, though we feel it should be given because of its general stimulant properties.

6. Reports have recently appeared (30), (27), concerning the effectiveness of intravenous PAM (Pyridine-2-aldoxine methiodide) as an antidote in cases of alkyl-phosphate poisoning. Synthetic alkyl-phosphates are used as remedies (isofluorophosphate (DFP)) or as insecticides (tetraethylpyrophosphate (TEPP)) parathion. This latter preparation is used extensively in Japan by workers spraying rice fields, who may in severe cases show respiratory depression and prolonged coma, together with other symptoms as a result of parathion poisoning. In these patients a low level of serum pseudocholinesterase is also observed. Previously treated with atropine, it has been shown that an intravenous injection of PAM (usual dose 1 Gm.) reverses some of the symptoms instantly, and results in reactivation of the pseudocholinesterase to normal levels. No side-effects have been reported so far following the administration of PAM, which may also be given orally.

Perhaps PAM is the specific antidote also in cases of respiratory paralysis following the use of muscle relaxants, associated with low serum pseudocholinesterase, and it is intended to try it out in any future examples of this condition that come to our notice.

SUMMARY

Prolonged apnoea following suxethonium (Brevidil "E"), a short-acting muscle relaxant used for modifying electrically induced convulsions, does not appear to have been reported previously, though numerous examples of this complication have been described following suxamethonium (succinylcholine, "Scoline"), a closely related chemical compound.

Three cases of prolonged apnoea following suxethonium (1 hour, 45 minutes and 10 minutes) are described, all of whom showed low serum pseudocholinesterase levels.

The causes of this complication are discussed in the light of this finding, together with the mode of action of the depolarizing group of relaxants to which suxethonium belongs. Other factors must be responsible for prolonged apnoea when the pseudocholinesterase levels are normal, and these are discussed.

The importance of apnoea following modified E.C.T. is stressed, since it has been ascertained that at approximately 85 per cent. of hospitals that give E.C.T. muscle relaxants are used. Suxethonium is used at 22 per cent. However, Consultant Anaesthetists are present at only 50 per cent. of mental hospitals during routine electroplexy sessions.

Some points concerning methods of prevention and treatment of prolonged apnoea are given.

It has been suggested that PAM (Pyridine-2-aldoxine methiodide) which successfully reverses some of the symptoms of alkyl-phosphate (Parathion) poisoning, and restores the serum pseudocholinesterase to normal levels, might act as a specific antidote in these cases of prolonged apnoea, and it is intended to try out this preparation in the future.

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NOTE

The Brevidil "E" used in these cases was supplied by Messrs. May & Baker Ltd. in ampoules containing 150 mg. of the total salt (100 mg. of the cation).

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A CLINICAL TRIAL OF COVATIN ON ELDERLY PATIENTS WITH ANXIETY AND RESTLESSNESS

By

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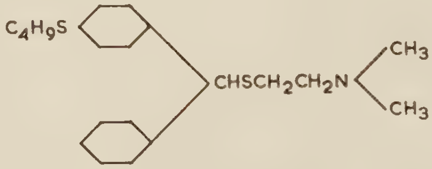
DIPHENHYDRAMINE hydrochloride has been found to have a sedative effect, and various modifications of it have been prepared in order to enhance its effect. A promising variant is Covatin*, which is p-butylthiodiphenylmethyl-2-dimethylaminoethyl sulphide hydrochloride; or, structurally:



The butylthio-substitution in the para-position is likely to provide increased sedation and to decrease toxicity. The sulphur in the centre chain may increase the sedative effect by making the compound a thio-ether. The 2-methyl in the amino group may further enhance the sedative action. As Covatin is only slightly related to other sedatives, a trial of its action was considered to be of special interest.

Its toxicity in mice has been found to be, as LD 50:

Intravenously		44	mg./kg.
Intraperitoneally		116	mg./kg.
Orally	about	1,500	mg./kg.

There were no changes in the red or white cell counts in rats eight hours after the administration of a single subcutaneous dose of 1 G./kg. Covatin. Nor was there a rise in the methaemoglobin percentage, when cats were given Covatin intravenously 5 mg./kg. as well as 50 mg./kg. subcutaneously.

Rats receiving orally 10 mg./kg. and 100 mg./kg. daily for 3 months were quite normal. This represents 20 times the maximum dose.

Earlier trials (Ellerman (1); Werenberg (2)) had given promising results with younger patients, so we considered it worth while to carry out a properly designed clinical trial on the elderly.

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* William R. Warner & Co.

THE INVESTIGATION

The patients were all women, aged between 60 and 80 years, all fairly stationary in their clinical conditions, and all suffering from a degree of restlessness, anxiety or tension, of a degree sufficient to demand some sedative. Otherwise they were not specially selected.

Each patient acted as her own control. For 14 days she was given one substance as one tablet t.d.s. (either Covatin 50 mg. or placebo). (Which was given was determined by chance, from a table of random numbers.) Then for 14 days she was given the other substance similarly. After this four weeks, another four weeks followed, similar except that now two tablets were given. Which was given first was also determined by chance, independently of how the first four weeks' chance had fallen.

A schedule for the substances and doses was drawn up, so that only the dispenser knew whether the patient was having drug or placebo. In particular, this knowledge was withheld with special care from the patient, and from those who assessed her clinical condition.

No patient had E.C.T. or any major treatment during the test; and though some patients needed mild sedation, the extra sedative was kept at the lowest possible dose and in most cases was unchanged throughout the test. A subsequent review of the protocols and the patients' medicine cards showed no reason to suspect any appreciable error from this factor.

Careful clinical records were kept of the patients' general behaviour and demeanour. In addition, a 26-question form was drawn up and was answered at the end of each week, for each patient, by the sister who had been looking after her. The questions were as follows:

1. Has the patient been restless?
2. Does she pick at herself?
3. Does she pick at her clothing?
4. Does she pluck the sheets?
5. Does she get in and out of bed restlessly?
6. Does she seem worried?
7. Does she sleep badly at night?
8. Does she have sedation at night?
9. Does she sleep during the day?
10. Is it difficult to attract her attention?
11. Is she unsteady on her feet?
12. Does she fall about at all?
13. Does she have any phobias?
14. Is she afraid in any way?
15. Is she startled easily?
16. Does she complain of noise or sudden noise?
17. Does she worry about her health or appearance?
18. Is she incontinent by night/day?
19. Does she mind being alone—night/day?
20. Does she make silly requests?
21. Is she hallucinated?
22. Is she excitable, generally, specifically?
23. Do her moods fluctuate quickly, i.e. hour to hour/day to day?
24. How does she react to other patients:
    (*a*) indifferent?
    (*b*) friendly?
    (*c*) hostile?
25. How does she react to Staff:
    (*a*) indifferent?
    (*b*) friendly?
    (*c*) hostile?
26. What does she say about herself:
    (*a*) disinterested?
    (*b*) forward looking—hopeful—hopeless?
    (*c*) backward looking?

It will be seen that most of the questions can be answered by a simple Yes or No. The sisters who undertook the scoring were specially instructed; they proved to be most helpful and conscientious. Their great personal knowledge of each patient made their judgments and answers specially reliable and sensitive.

One patient died during the test from an unrelated illness; one refused to continue. Seventeen patients were taken through the full procedure.

THE RESULTS

When the sequence was concluded, and the results reviewed, it was apparent that little effect was detectable. No general improvement had occurred, and no detectable change had taken place, either at low or high dosage, when the tablets were changed from placebo to drug or in the opposite direction.

No toxic or unpleasant effects, such as nausea or vomiting, occurred.

Some patients responded markedly to the commencement of the sequence of tablet-giving, but reference to the protocol showed that this effect was about equally likely to appear with the placebo or with the drug.

A more detailed study was made of the facts in the questionnaires. As these contained 17×8×26, i.e. 3,536 binary units of information, a statistical analysis of them provided a more sensitive indicator than could be obtained by direct observation. The questions were therefore classified according to whether the answer "Yes" indicated the presence of "anxiety". A count of the Yes's thus gave a measure of the patient's degree of anxiety over that week. Two weeks' scores added gave the score for anxiety while under the drug (or placebo); its difference from that for the next two weeks (on placebo or drug) gave an unbiased estimate of the change caused by the drug. This difference is that due to one tablet; the whole process repeated over the next four weeks' forms gave the change caused by the drug at a level of two tablets.

When the results were examined, tests of significance (the anti-chance odds) showed that there was no evidence that the drug, either at one tablet or two t.d.s., caused any detectable shift in the degree of anxiety.

The forms were then re-examined, and the questions selected according to whether they indicated "motor restlessness"; a similar analysis was then applied. (The result is not, of course, independent of the test on anxiety.) Again no significant effect could be found.

Finally the questions were selected for those that indicated the patients' "sleepiness", and a similar set of scores found and tested. Again no significant effect could be found. It seems therefore, that in this series, Covatin had no marked sedative effect.

SUMMARY

The effect of Covatin was tested on seventeen elderly patients, all women, who, over eight weeks, received either a placebo or Covatin at 50 mg. or at 100 mg. t.d.s. The response of each patient was measured weekly on a 26-item questionnaire designed to estimate her degree of anxiety, and restlessness, and sleepiness. Neither patient nor those assessing her condition could know whether she was, at that time, receiving drug or placebo.

The results showed no evidence for any significant effect. No complications or toxic symptoms occurred.

ACKNOWLEDGMENTS

We would like to express our indebtedness to Messrs. William R. Warner & Company, who generously provided the Covatin and the placebo, and who were helpful in many ways.

Our thanks are also due to Sisters Edwards, Rees, Duffield and James who undertook the extra work with enthusiasm and care.

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QUANTITATIVE COMPARISON OF CLINICAL EFFECTIVENESS OF CHLORPROMAZINE AND PROMAZINE

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A DOUBLE-BLIND study of chlorpromazine and promazine was conducted to measure their comparative therapeutic effectiveness on a short-term basis at medium dosage levels, such as might be used for out-patient purposes. The patients chosen, however, represented a more therapeutically challenging group of hospitalized acute schizophrenics in whom a dramatic and convincing effect might offer an evaluation of comparative response.

METHOD

Forty-five newly admitted, acute schizophrenics, following a period of admission evaluation were assigned alternatively to three different groups (G, X, and V) in a double-blind comparison. Each patient was evaluated independently by a psychiatrist and two members of the nursing service on different shifts. The patients' average age was 31·2 years and equivalent from group to group. Laboratory work included CBC and alkaline phosphatase. Pertinent physical findings including routine blood pressures were recorded.

Members of the three groups received in identical oral capsules either a placebo (G), chlorpromazine (X), or promazine (V). The dosage of each was raised gradually over a 10-day period to a level of 400 mg./day of chlorpromazine, and continued at this level for three weeks. Behavioural and laboratory studies were repeated at the end of this period.

Six patients (two from G, one from V, and three from X) were given parenteral reserpine (5 mg. I.M.) during periods of unmanageable hyperactivity. Number of injections varied from 1 to 9 with four of the six patients not receiving more than 2 injections. The necessity for this seemed to be related more to the patient's pre-treatment level of excitation than to the experimental drug used.

The only side-reaction observed during the experimental treatments was a mild, generalized erythematous rash occurring on the 21st day of treatment in one patient receiving drug X (chlorpromazine).

Although 45 patients initially were entered into the study, only 37 could be followed to conclusion. The other eight patients were dropped because of medical illness, unplanned discharge or transfer, and similar circumstances beyond our control but without bias to the study.

Every patient was initially rated (pre-treatment) and then re-rated (post-treatment) by his psychiatrist and each of two ward nurses for the presence and intensity of (a) *Delusions*, including ideas of reference, (b) *Hallucinations*, and (c) *Disturbed Behaviour*. The latter, "Disturbed Behaviour," was subdivided into two ratings: one for the continuum ranging from "Normal" to "Agitated or Assaultive," the other for the continuum from "Normal" to "Severe Withdrawal." In all ratings the higher score signified greater pathology.

These three independent ratings for each patient were then additively combined pre- and again post-treatment to yield an overall pathology score which incorporated the three major dimensions of pathology (*Delusions*, *Hallucinations*, *Disturbed Behaviour*).

An overall pathology score was thus derived for each patient prior to the experimental medication (pre-) and then after three weeks of that medication at maximum dosage 400 mg./day (post-). This score ranged from no pathology at 0 to maximum pathology at 30.

RESULTS

The mean overall pathology scores for the G, V, and X groups pre- and post-treatment are presented in Table I, together with the results of their significance tests for the differences pre- to post-.

TABLE I

Mean Pathology Score and Degree of Improvement Pre- to Post-Treatment Under Each Method

Treatment Method		Mean Pathology Score	Mean Improvement*	Per cent. Improvement	*t*	*p*
G (Placebo)	Pre-	10·99				
(N=13)	*vs*		3·11	28·3	3·05	·02†
	Post-	7·88				
V (Promazine)	Pre-	10·95				
(N=14)	*vs*		3·59	32·8	3·16	·01†
	Post-	7·36				
X (Chlorpromazine)	Pre-	11·73				
(N=10)	*vs*		6·43	54·7	4·87	·001†
	Post-	5·31				

* The greater the degree of improvement, the greater the drop in pathology.
† Statistically significant at the indicated level.

Significance tests were also conducted for the initial mean scores (pre-) among G, V, and X, to ensure that the mean degree of initial pathology was similar for the patients entered into each of the three methods (G, V, or X). Results of these tests showed *no* significant differences in mean initial pathology. The initial mean degree of pathology was equivalent for the patients entered into each of the three treatment methods (G, V, or X).

Examining Table I, we find that significant improvement occurred under each of the three treatments. This was consistent with expectation since all patients initially were in a psychotic state and numerous remissions might be expected in consequence of the multiple treatment modalities—equitably administered to the three groups—which are always provided to hospitalized psychiatric patients. These included the various rehabilitation, psychological, and recreational therapies, but of course they excluded somatic or pharmacodynamic therapies for these patients. It is notable, however, that the *per cent.* improvement was greater for chlorpromazine than for promazine or placebo.

A more important question is therefore this: Does any one of these methods, G, V, or X, yield a statistically significant *greater degree* of improvement (pre- to post-treatment) than do the other two?

To answer this question improvement scores (decrease in pathology pre- to post-treatment) were calculated for every patient under each of the three treatment methods. The mean improvement scores then were compared among G, V, and X. These results are reported in Table II. Since improvement was the converse of pathology, the total possible gradient of improvement scores ranged from no improvement at 0 to full recovery at 30. The greater the improvement score, the greater the decrease in pathology.

TABLE II

Significance of the Differences in Mean Improvement Scores Among the Three Methods

Treatment Method	Mean Improvement	*t*	*p*
G (Placebo)	3·11		
vs		·47	·40
V (Promazine)	3·59		
V (Promazine)	3·59		
vs		5·34	·001*
X (Chlorpromazine)	6·42		
G (Placebo)	3·11		
vs		3·00	·001*
X (Chlorpromazine)	6·42		

* Statistically significant at the indicated level.

The degree of improvement under X was markedly superior to that under V and under G. V was somewhat more effective than G but this was statistically insignificant. It is therefore concluded that, within the method and limits of this experiment, chlorpromazine (X) was significantly more effective than promazine (V) and placebo (G) in reducing overall pathology. No significant difference was present in the comparative effectiveness of promazine as against the placebo.

Conclusions drawn from this study must be limited by the conditions under which it was conducted. It does seem however, that milligram for milligram over a one-month period, a significantly greater number of acute schizophrenic patients can be rendered less psychotic with chlorpromazine than with promazine or hospitalization alone. No conclusion can be drawn about the relative incidence of side-effects, due to the briefness of the study and the small patient sample.

SUMMARY

A double-blind study of chlorpromazine and promazine, together with placebo, was conducted to measure their comparative therapeutic effectiveness at medium dosage levels. The subjects were newly hospitalized, acutely schizophrenic patients. Over-all pathology scores were compared before and after treatment under each method. Within the procedures of this study, chlorpromazine was significantly more effective than either promazine or placebo in alleviating mental pathology. No significant difference was noted between promazine and placebo.

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A STUDY ON SERUM COPPER LEVEL IN 20 ACUTE SCHIZOPHRENICS*

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Blood copper had been known to be increased in pregnancy, chronic infections and neoplasms. Since the publication of Ullrich in 1942 (9), who found an increased copper level in the blood of schizophrenics, the hypercupraemia has been recognized as possibly significant and pathognomonic in this disease. Therefore during the following years a few investigators studied this aspect and also tried to prove the implications of this fact.

Brenner and Breier (3) and Bischoff (2) found a high blood-copper level in schizophrenics.

It was found that 96 per cent. of the blood copper is bound to an α_2–globulin, named coeruloplasmin (6). This copper globulin has been recognized as the enzyme in serum, which catalyses epinephrine oxidation, at least in vitro (7). Leach *et al.* (7) also found a significantly high blood-copper content bound to coeruloplasmin in schizophrenics as compared with mentally healthy controls. This high copper level and simultaneously elevated serum amine-oxidase might be of some importance in epinephrine metabolism, as aberrations in the breakdown of epinephrine seem to make possible the liberation of toxic and hallucinogenic substances as adrenochrome and adrenolutin.

Lately, however, studies have been undertaken by Aprison *et al.* (1), Frohman *et al.* (5) and Scheinberg (8), who could not find any significant difference in blood copper or coeruloplasmin content in schizophrenics as compared with normals.

Stimulated by the investigation in this subject and by the possible important implications of its results on further enzymatic research, we examined the serum-copper content of a small number of schizophrenic patients.

Material

We examined the blood of 20 schizophrenic in-patients, who had not had any treatment yet, either at their home or in the hospital after admission. Their respective ages were between 22 and 45 years. As this is a country of constant immigration it might be of interest to state their various countries of origin: born in Israel were 4; from Poland 4; Germany 1; Bulgaria 1; Roumania 1;

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Chile 1; Morocco 1; The Yemen 2; Syria 1; Afghanistan 1; Turkey 1; Iraq 2.

Of the 20 schizophrenics, 10 were of the paranoid type, 3 catatonics, 2 hebephrenics, 3 simplex and 2 of the circular form. 10 control cases were taken from members of the hospital staff, belonging to the same age group of from 22 to 45 years.

METHOD

Blood was taken by syringes and needles, which had been previously treated by bi-distilled water.

Serum proteins were precipitated by trichloracetic acid. The clear filtrate was treated with sodium diethyl-dithiocarbamate and the amount of copper determined by the method of Cartwright *et al.* (4) using a photo-electric colorimeter (Lumetron model 402-E). The concentration of copper in normal serum by this method varies between 80 and 160 mcg per 100 ml. of serum.

RESULT

SCHIZOPHRENICS:		*Copper content, expressed in mcg. per 100 ml.*			
(a) Paranoids:	(1) 120.	(2) 164.	(3) 100.	(4) 72.	(5) 106.
	(6) 177.	(7) 104.	(8) 160.	(9) 130.	(10) 115.
(b) Catatonics:	(11) 132.	(12) 120.	(13) 125.		
(c) Hebephrenics:	(14) 144.	(15) 130.			
(d) Simplex:	(16) 138.	(17) 200.	(18) 110.		
(e) Circular:	(19) 126.	(20) 96.			

The average of the serum copper level of all schizophrenics was 128 mcg/100 ml. serum.

Normal controls ranged between 66 to 142 mcg/100 ml., with an average of 107·5 mcg/100 ml. serum.

Only 3 samples of schizophrenic blood were at the upper limit obtainable by this technique or showed only an insignificant increase.

The Standard Deviation (SD) of the schizophrenic copper level was 28·5, that of the controls was 20·15. Therefore the difference in variability was a very slight and insignificant one.

Although this pilot study includes only a relatively small number of cases, we are inclined to believe, as other authors lately, that there is no significant hypercupraemia to be found in schizophrenics. Therefore, the copper level, cannot be used as an auxilliary diagnostic test, nor does it seem to show any connection with aberrant epinephrine metabolism, as far as the latter is concerned with the enzymatic breakdown by the amine oxidase coeruloplasmin.

SUMMARY

Blood samples of 20 acute cases of Schizophrenics were examined for their copper content. No significant increase in serum-copper level as compared with normal controls could be found by this method.

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Bibliography and Epitome

VOL. 57 ACTA NEUR. PSYCHIAT. BELG. NOVEMBER, 1957

The Obersteiner Institute and the Destiny of Modern Neuropathology. *Van Bogaert, L.* 861
Kleptomania. *Massiou-Verniory, L.* 869
The Titre of 17-ketosteroids in Various Masculine Seropathies. *Massiou-Verniory, L., et al.* 890
Anatomo-clinical Contributions to the Study of Huntington's Chorea. *Façon, E., et al.* 898
Progressive Subacute Hyperkinetic Encephalitis in Children in Bulgaria. *Usunoff, G., et al.* 913
The Treatment of Paralysis Caused by Disseminated Sclerosis and Poliomyelitis with Amnion. *Mieczyslaw, S.* 923
Schizophasia and Cybernetics. *dal Bianco, P.* 937
The Psychological Basis of Schizophrenic Language. *Kreitler, M.* 950
Inhibition of Language and Mutism in Psychotherapy. *Tolentino, I.* 955
Affects and Frustrations seen in the Drawings of Schizophrenics. *Sechehaye, M.* 972
"Portmanteau Words" of Lewis Carroll (or "Mots Valises") during Schizophrenia. *Ferdiere, G.* 993
The Psychopathology of Verbal Expression. *Stuchlik, J.* 1004
The Psychopathology of Expression, Plastic, Mimic and Pictural "Neomimisms" and "Neomorphisms". *Bobon, J.* 1031

VOL. 58 1958

The Mechanism of Faradic Epilepsy. *Bremer, F.* 5
Some Observations on "Genetic" Epilepsy. *Cordier, J.* 10
Some Remarks on a Verified Epileptic State. *Cordier, J.* 26
Electro-clinical Correlations in 300 Cases of Convulsions in Children. *André-Balisaux, G., and Gonsette, R.* 31
Hyperthermic Convulsions in Children. *Radermecker, J.* 50
Infantile Encephalopathy with Epilepsy and Death during Furunculosis. *Radermecker, J.* 56
Inter-fit Psychotic States in Epilepsy. *Cordier, J.* 95
Anatomo-clinical Facts on Epilepsy. *Deschamps, A., et al.* 105
A Spinocerebellar Degeneration with Unilateral Involuntary Movements of Athetoid Type in a Feeble Epileptic. *Colle, G.* 114
Remarks on a Temporal Epilepsy with a Small Unrecognised Tumour of the Amygdaloid Region, etc. *Roussel, J.* 124
The Treatment with ACTH of 21 Cases of Hypsarrhythmia of Gibbs. *Dusaucy-Bauloye, S. L.* 130
Synchronous, Bilateral, EEG Paroxysms. *Meurice, E.* 155
Hypersexuality in Tumours of the Temporal Lobe. *van Reeth, P. C.* 194
The EEG during E.C.T. Convulsions. *Piette, Y.* 219
Experimental Arteriography of Brain Vasomotor Centres. *van den Bergh, R.* 231
Syndrome of the Cauda Equina with Herniated Disc. *Hasaerts, R., and Brihaye, J.* 247
Relations Between Syringomyelia and Familial Acropathy. *Ionesco, I., et al.* 260
A Senile Cerebellar Atrophy. *Henneaux, J.* 270
Histology of Some Physiopathological Aspects of Human Poliomyelitis. *Laruelle, L., and Reumont, M.* 297
The Borders of Schizophrenia. *Errard, E.* 356
Three Cases of Actinomycotic Cerebral Abscess. *Brucher, J. M., et al.* 372
Clinico-statistical Study of 93 Cases of Infiltrating Glioma of the Brain-stem. *Arseni, C., and Goldenberg, M.* 404
The Syndrome of Vascular Insufficiency of the Basilar Arterial System. *Poser, C.* 420
Diagnostic Value of Carotid Angiography in the Syndromes of Cranial Hypertension without Localising Signs. *Poser, C.* 436
Anatomo-clinical Aspects of Certain Encephalitides Wrongly Classified, etc. *Draganesco, S., et al.* 495
The Influence of Rapid Labour without Pain on the Cerebral State of the Infant. *Goussis, A.* 512
A Case of Encephalitis from Flue Virus "Singapore 57". *Dupont, E., and Keefer, W.* 532

VOL. 33 ACTA PSYCHIAT. NEUR. SCAND. 1958

Some Experiences in Abreaction by Ether Inhalation. *Askevold, F.* 257
Vestibulo-ocular Reflex Disorders of the Anterior Internuclear Paralysis Type in Supratentorial Space-taking Lesions. *Ethelberg, S., and Vaernet, K.* 268
The Significance of Electrophoretic Investigation in Inflammatory Diseases of the Nervous System. *Hanzal, F., and Vymazal, J.* 283

Etiological Factors of Cryptogenic Epilepsy, Especially the Importance of Mother's Age at the Birth of the Patient and the Patient's Place in the Series of Siblings. *Hertoft, P., et al.* .. 296

Psychiatric Investigations of Patients in a Medical Department. *Helsborg, H. C.* .. 303

Pigmentary Retinal Lipoid Neuronal Heredodegeneration. *Hoffman, J.* .. 336

Course and Prognosis in Spontaneous Occlusions of Cerebral Arteries. *Lindgren, S. O.* 343

Fibre Tracts in the Cord in Cat. *Verhaart, W. J. C., and van Beusekom, G. T.* .. 359

Clinical and Genetic Studies on Benign Schizophreniform Psychoses Based on a Follow-up. *Welner, J., and Strömgren, E.* .. 377

VOL. 33 ACTA PSYCHIAT. NEUR. SCAND. SUPPT. 1958

Mental Symptoms in Temporal Lobe Epilepsy and Temporal Lobe Gliomas. *Bingley, T.* .. Suppt. 120

Mental and Physical Illness Among Paupers in Stockholm. *Inghe, G.* .. Suppt. 121

A Psychometric Study of Identical Twins Discordant for Closed Head Injury. *Denckner, S. J., and Löfning, B.* .. Suppt. 122

A Follow-up Study of 128 Closed Head Injuries in Twins Using Co-Twins as Controls. *Dencker, S. J.* .. Suppt. 123

The Mothers of Schizophrenic Patients. *Alanen, Y. O.* .. Suppt. 124

A Study of Schizophrenia in the Male. *Johanson, E.* .. Suppt. 125

VOL. 62 AM. J. MENT. DEFIC. JANUARY, 1958

Programming for the Mentally Retarded as Proposed by the University and State Department of Health. *Deisher, R. W., and McNellis, E. C.* .. 579

The Normal Development of the Mentally Retarded Child: Planning a Federal Program. *Duhl, L. J.* .. 585

Delegating Administrative Responsibilities to Ward Service or Cottage Personnel. *Shawver, L. E.* .. 592

Professional Nursing Personnel and Their Functions. *Kassler, R.* .. 597

Selecting the Psychiatric Aide. *Bissett, A.* .. 601

The Role of the Aide in Administration. *Pockoski, W. J.* .. 603

Religious Training of the Mentally Retarded at Caswell Training School. *Hurt, J. M.* 607

Developing a Curriculum for the Blind Retarded. *Tretakoff, M. I., and Farrell, M. J.* 610

A Curriculum for the Severely Mentally Retarded. *McCaw, W. R.* .. 616

A Study of the Metropolitan Achievement Test Results of Educable Girls in Residence at the Rome State School. *Shachoy, G. R.* .. 622

Providing Occupational Readiness for Young Mentally Deficient Children of the Non-Familial Type. *McCartney, L. D.* .. 625

A Study of the Incidence of Mongolism in Relation to the Fluoride Content of Water. *Berry, W. T. C.* .. 634

Some Psychiatric Problems Related to Mental Retardation. *Forbes, L. M.* .. 637

Vitamin A Absorption and Other Blood Composition Studies in Mongolism. *Sobel, A. E., et al.* .. 642

An Evaluation of the Nutritive Supplement Dietall. *Berman, H. H., and Noé, O.* .. 657

The Effects of Tranquilizing Medication (Reserpine) on Behavior and Test Performance of Maladjusted, High-Grade Retarded Children. *Rosenblum, S., et al.* .. 663

Experimental Psychology and Psycho-asthenics. *Cassel, R. H.* .. 672

The Incidence of Hearing Loss in Mentally Retarded Children. *Kodman, F., Jr.* .. 675

Traveling Clinics and the Retarded Child: The North Carolina Story. *Griffin, D. P.* 679

Need for Achievement as Related to Brain Injury in Mentally Retarded Children. *Tolman, N. G., and Johnson, A. P.* .. 692

The "Pointing Modification" of the Revised Stanford-Binet Intelligence Scales, Forms L. and M, Years II Through VI: A Report of Research in Progress. *Katz, E.* .. 698

An Investigation of the Use of the D.A.P. in the Diagnosis of Homosexuality in Mentally Deficient Males. *Vilhotti, A. J.* .. 708

Unmet Needs of the Mentally Retarded in the Community. *Begab, M. J.* .. 712

MARCH

Convention Program .. 759

Mental Deficiency—1957. *Whitney, A. E.* .. 771

A National Program for Mental Retardation: A New and Better Beginning. *Douglass, J. H.* .. 777

Evaluating Vocational Aptitudes and Characteristics of Mentally Retarded Young Adults in an Industrial-Agricultural Workshop. *Ferguson, R. G.* .. 787

Academic Requirements of Jobs Held by Educable Mentally Retarded in the State of Connecticut. *Young, M. A.* .. 792

Study of Therapeutic and Functional Values of Hearing Aids for the Mentally Handicapped. *Hartman, B.-T.* .. 803

The Physical, Personality, and Academic Status of Children Who are Mentally Retarded Attending Special Classes as Compared with Children Who are Mentally Retarded Attending Regular Classes. *Blatt, B.* .. 810

Use of Mentally Retarded Children on Television Programs as a Medium of Public Relations. *Howard, C. J.*	819
Coordination of Client's Experiences from School to Occupational Center. *Goldberg, I. I.*	823
A Survey of the Literature on Curriculum Practices for the Mentally Retarded. *Wolk, S. M.*	826
Patterns of Discovery in the Genetics of Mental Deficiency. *Allen, G.*	840
Britain Takes a New Look at Mental Illness and Mental Deficiency. *Tarjan, G.*	850
A Clinical Evaluation of Psychopharmacological Agents in the Management of Disturbed Mentally Defective Patients. *Rudy, L. H., et al.*	855
Oligophrenia in Pseudohypoparathyroidism (The Seabright-Bantam Syndrome). *MacGillivray, R. C.*	861
An Interdisciplinary Personnel Training Program in a Specialized Clinic for Retarded Children. *Slobody, L., et al.*	866
Learning and Mental Deficiency. *McPherson, M. W.*	870
A Comparison of the Wechsler Adult Intelligence Scale and Wechsler-Bellevue II with Mental Defectives. *Light, M. L., and Chambers, W. R.*	878
Studies in Activity Level: I. Simple and Complex Task Performance in Defectives. *Foshee, J. G.*	882
A Proposed Role for the School Psychologist: Counseling Parents of Mentally Retarded Children. *Ruzicka, W. J.*	897
Diagnostic Classification, Intellectual Characteristics and Parental Occupations of Five Hundred Severely Deficient Institutionalized Males. *Murphy, M. M.*	905
Mosaic Differences in Non-institutionalized Retarded Children. *Carr, G. L.*	908
The Psychological Development of the Mentally Retarded Child: A Training Course for Attendants. *Graves, W. S.*	912
The Performance of Mentally Retarded Children on the Weigl-Goldstein-Scheerer Color Form Sorting Test. *Halpin, V. G.*	916
A Social Theory of Mental Deficiency. *Dexter, L. A.*	920
A Technique for an Area Study Pertaining to Mental Deficiency. *Lernerd, M. B.*	929
Training and Placement of Defectives in a Residential School Through the Division of Vocational Rehabilitation. *Russell, F. W., and Mulcahy, R. M.*	935

MAY

Editorial. *Duhl, L. J.*	979
Introduction: Historical Data. *Powers, G. F.*	981
Implications of the Reports for Future Progress in Mental Retardation. *Shannon, J. A.*	986
The Prevention of Mental Retardation: A Survey of Research. *Masland, R. L.*	991
Psychological and Cultural Problems in Mental Subnormality: A Review of Research. *Sarason, S. B., and Gladwin, T.*	1115

VOL. 63 JULY

Introduction of the President, Prof. Chris J. DeProspo. *Hungerford, R. H.*	2
Presidential Address—"Crossroads". *DeProspo, C. J.*	3
Mental Deficiency (1957) Supplemental Abstracts. *Whitney, E. A.*	13
Towards More Effective Use of the Term Mental Deficiency. *Jordon, T. E.*	15
Language Training of Low-Grade Mongoloid Children. *Kolstoe, O. P.*	17
Basic Issues Concerning the Education of Children with Cerebral Defects. *Halpin, V.*	31
Editorial. *Masland, R. L.*	38
The Medical School Phase of the Education of a Physician. *Caughey, J. L., Jr.*	29
Withdrawals from License in Mental Deficiency. *Craft, M.*	47
Some Cultural and Personality Aspects of Mental Retardation. *Ruess, A. L.*	50
Techniques and Expressive Therapy Integrated into the Treatment of Mentally Retarded Children. *Gondor, E. I., and Levbarg, M.*	60
Small Group Observation as a Diagnostic Technique. *Lederman, D. G.*	64
Performance of Brain-Injured and Familial Mentally Deficient Children on Visual and Auditory Sequences. *Hunt, B., and Patterson, R. M.*	72
Observations Concerning Counseling with Parents of Mentally Retarded Children. *Mahoney, S. C.*	81
Some Different Emphases in the Role of the Social Worker in a Workshop for Mentally Retarded Adolescents and Young Adults. *Nitzberg, J.*	87
Social Work with the Mentally Retarded in Maryland's State Institutions. *DeWitt, H. B.*	96

SEPTEMBER

Call for Papers and Symposia. *Rosebrook, W., and Simmons, P.*	197
Suggestions for Contributors to the American Journal of Mental Deficiency. *May, B.*	200
Recommended Criteria to be Used by Division Editors in Approving Articles for Publication. *Rosenzweig, L. E.*	203
Schools for Exceptional Children. Reprinted from "Education in the U.S.S.R." Bulletin 1957, No. 14.	204
Mental Deficiency in Poland and the U.S.S.R. *Wollen, W.*	205
Terminology and the Classification of Mental Retardation. *Heber, R.*	214

Opportunities for Training and Research Through Cooperation of Universities and Residential Facilities for the Retarded. *Hindman, D. A.* 220

An Analysis of the Objectives for the Education of Children with Retarded Mental Development. *Stevens, G. D.* 225

A Review of Research in Speech and Language Development of the Mentally Retarded Child. *Harrison, S.* 236

Levels of Abstraction in Vocabulary Definitions of Mentally Retarded School Children. *Badt, M. I.* 241

Some Relationships Between Abstraction and Word Meaning in Retarded Adolescents. *Griffith, B. C., and Spitz, H. H.* 247

A Teaching Sequence for the Training of Visual and Motor Perception. *Jolles, I.* 252

Perceptual Processes in Normal Development, Brain Injury and Mental Retardation. *Zuk, G. H.* 256

Exploring the Relationship Between the Teacher's Attitude and the Overt Behavior of the Pupil. *Harris, L. M.* 260

A Large Scale Music Therapy Program for Institutionalized Low Grade and Middle Grade Defectives. *Murphy, M. M.* 268

Programs for Severely Mentally Retarded Pupils. *Salvin, S. T.* 274

Contributions of Projective Tests to Problems of Psychological Diagnosis in Mental Deficiency. *Molish, H. B.* 282

Low Level of Intellectual Functioning and Dissocial Behaviour in Children. *Frankenstein, C.* 294

Relationship Between Intelligence and Skin Conductance. *Ellis, N. R., and Sloan, W.* 304

Changes in Terman Merrill I.Q.s of Mentally Retarded Children. *Collmann, R. D., and Newlyn, D.* 307

A Comparison of the Wechsler Intelligence Scale for Children and the Arthur Adaptation of the Leiter International Performance Scale with Mental Defectives. *Alper, A. E.* 312

The Evaluation of Conceptual Ability as Related to the Academic Achievement of the Educable Mentally Handicapped. *Cutts, R. A.* 317

The Significance of Heterozygosity for Hereditary Metabolic Errors Related to Mental Deficiency (Oligomentia). *Lippman, R. W.* 320

Ascorbic Acid and Cholesterol Studies on Women in an Institution for the Handicapped. *Potgieter, M., et al.* 325

The Use of Reserpine and Chlorpromazine in Disturbed Mentally Deficient Patients. *Wardell, D. W., et al.* 330

The Relation of Mongolian Stigmata to Intellectual Status. *Gibson, D., and Gibbins, R. J.* 345

Impressions of the Social Problems of Mental Retardation in the United States of America. *Thomas, D. H. H.* 349

Work Procurement and Job Production. *Dubrow, M.* 355

Report to the Association: Status of the AAMD Project "Technical Planning in Mental Retardation". *Nisonger, H.* 360

VOL. 28 AM. J. ORTHOPSYCHIAT. JANUARY, 1958

Segregation-Integration: Some Psychological Realities. Round Table, 1957. *Beck, S. J., et al.* 12

Psychoanalytic Concepts and Principles Discernible in Projective Personality Tests. Workshop, 1956. *Piotrowski, Z. A.*

1. Freud's Psychoanalysis and Rorschach's Perceptanalysis. *Piotrowski, Z. A.* 36
2. Psychoanalytic Principles Discernible in Projective Testing. *Bellak, L.* 42
3. Mechanisms of Defense in Schizophrenic Reaction Types as Evaluated by the Rorschach Test. *Molish, H. B., and Beck, S. J.* 47
4. Application of Psychoanalytic Principles to the Rorschach Perceptanalysis in a Longitudinal Study. *Fries, M. E.* 61
5. The Psychoanalytic Interpretation of Dreams and Projective Techniques. *Fromm, E.* 67
6. The Predictive Significance of Fantasy. *Symonds, P. M., and Jensen, A. R.* 73

The Emotional Significance of Acquired Physical Disfigurement in Children. *Watson, E. J., and Johnson, A. M.* 85

Renunciation and Denial in Learning Difficulties. *Sperry, B., et al.* 98

The Mental Health Implications of Social Legislation. *Schottland, C. I.* 112

Special Problems in the Consultation Function of Child Guidance Clinics. *Zwick, P. A.* 123

Methods and Techniques of Consultation. *Abramovitz, A. B.* 126

Delinquency—A Laboratory for Public Health Psychiatry. *Peck, H. B.* 134

The Body Image of the Juvenile Delinquent. *Kaufman, I., and Heims, L.* 146

Relationships Between Maternal Attitudes and Child Personality Structure. *Altman, C. H.* 160

A Study of the Relationship of Early Menarche to Ego Development. *Rafferty, F. T., and Stein, E. S.* 170

Emotional Reactions to Medical Procedures and Illness in a Hospital Child Psychiatry Unit. *Sutton, H. A., et al.* 180

The Treatment of a Runaway Adolescent Girl Through Treatment of the Mother. *Wylie, D. C., and Weinreb, J.*	188
Reactions of Mothers of Twins in a Child Study Group. *Plank, E. N.*	196

APRIL

Basic Principles of Child Rearing in the Soviet Union: Firsthand Impressions of an American Observer. *Alt, H.*	223
The Mental Health of a Group of Russian Displaced Persons. *Hanfmann, E., and Beier, H.*	241
Psychiatric Consultation in Residential Treatment. Workshop, 1957. *Greenberg, H. A.*	
1. The Director's View. *Bettelheim, B.*	256
2. The Consultant's View. *Perkins, G. L.*	266
3. The Psychologist's View. *Wright, B.*	276
4. The Child Care Worker's View. *Riley, M. J.*	283
5. Discussion. *Adland, M.*	289
The Structured Ward: Research Method and Hypothesis in a Total Treatment Setting for Schizophrenia. *Scher, J. M.*	291
A Study of Patterns of Interaction of Families of Schizophrenic Children in Residential Treatment. *Behrens, M. L., and Goldfarb, W.*	300
The Changing Patterns of Women's Work: Some Psychological Correlates. *Ginzberg, E.*	313
Changing Concepts in the Care of the Aged. Workshop, 1957. *Grossman, B. L., et al.*	322
The Mental Health Board and Its Role in the Community. *Lemkau, P. V.*	343
Psychotherapy with the Nonreflective Aggressive Patient. *Morse, P. W.*	352
Signs of Disturbance in the First Year of Life. *Brody, S.*	362
The Significance of Atypical Postural and Grasping Behavior During the First Year of Life. *Fischer, L. K.*	368
Psychiatric Study of Mentally Retarded Children of Preschool Age: Report on First and Second Years of a Three-Year Project. *Woodward, K. F. et al.*	376
Hysteria in Childhood. *Proctor, J. T.*	394
The Restitutive Process in Children Following Radical Separation from Family and Culture. *Rathbun, C., et al.*	408
Psychiatric Observations of Children with Cleft Palate. *Tisza, V. B., et al.*	416
Inversion and Homosexuality. *Brown, D. G.*	424

JULY

Basic Science and the Future of Orthopsychiatry. *Lourie, R. S.*	445
The Mental Health Aspects of the Peaceful Use of Atomic Energy. *Fremont-Smith, F.*	456
Multidisciplinary Methods in Psychiatric Research. *Rioch, D. McK.*	467
Further Explorations of the "Six Schizophrenias". *Molish, H. B., and Beck, S. J.*	483
Psychiatric Teaching Programs in Pediatric Settings. Workshop, 1957. *Work, H. H., et al.*	506
Behavior Research in Collective Settlements in Israel:	
1. Editorial Statement. *Karpe, R.*	547
2. Collective Education in the Kibbutz. *Golan, S.*	549
3. The Development of the Young Child in a Collective Settlement. *Winograd, M.*	557
4. The American Social Scientist in Israel: A case Study in Role Conflict. *Rosenfeld, E.*	563
5. Some Problems of Research in Israeli Settlements. *Schwartz, R. D.*	572
6. Infants and Children under Conditions of "Intermittent" Mothering in the Kibbutz. *Rabin, A. I.*	577
7. The Study of Kibbutz Education and Its Bearing on the Theory of Development. *Rapaport, D.*	587
Group Gradients in Delinquency Potential and Achievement Scores of Sixth Graders. *Dinitz, S., et al.*	598
A Type of Predelinquent Behavior. *Josselyn, I. M.*	606
The Psychodynamics of Male Adolescent Delinquency. *Falstein, E. I.*	613
Initiating Therapy with Adolescents. *West, B., and Rafferty, F. T.*	627
The Relation of Motility to Boys' Learning Problems. *Sperry, B., et al.*	640
Implications of a Therapeutic Process in Evaluations by Psychiatric Teams. *Modlin, H. C., et al.*	647
Notes on Deprivations of the Privileged. *Rapoport, R. N.*	656

VOL. 115 **AM. J. PSYCHIAT.** JULY, 1958

Presidential Address. *Solomon, H. C.*	1
Harry Caesar Solomon, M.D., President—1957-58. A Biographical Sketch. *Sleeper, F. H.*	10
*The Amygdalae and Behavior. *Masserman, J. H., et al.*	14
Characteristics of Post-Partum Mental Illness. *Madden, J. J., et al.*	18
Diagnostic and Personality Testing in Clinical Psychology. *Payne, R. W.*	25
Subjective Reports of Lysergic Acid Experiences in a Context of Psychological Test Performance. *Cohen, S., et al.*	30

Some Conceptual Tendencies in the Psychosomatic Movement. *Bandler, B.* 36
The Correlation of Bilateral Occipital Slow Activity in the Human EEG With Certain Disorders of Behavior. *Cohn, R., and Nardini, J. E.* 44
Bilateral Adrenalectomy in Chronic Schizophrenic Patients: 6 Years After. *Apter, N. S.* 55
Object Relations Therapy in Schizophrenic States. *Azima, H., et al.* 60
Mind, Matter and Brain. *Roth, N.* 63
Considerations of the Utility of a Drug (Mepazine) in a Therapeutic Milieu. *Davis, F. H., and Weidorn, W. S.* 66
The Incidence of Hospitalized Mental Illness Among Religious Sisters in the U.S. *Kelley, M. W.* 72

The Amygdalae and Behaviour

The individual and social behaviour of 15 cats and 18 rhesus monkeys was recorded and analysed during a control period of from 3 to 15 months, during which the animals were also trained to solve increasingly complicated problems. Adaptational conflicts were used to induce persistent experimental neuroses in 3 kittens, 2 cats, 3 young monkeys and 8 adult ones. The amygdalae were then totally ablated, with some injury to the surrounding areas, in 6 of the cats and all of the monkeys; in the remaining 9 cats only the lateral or the medial amygdalae were removed.

Post-operatively, the cats exhibited: (1) no general amnesia for previous learning; (2) only mild amelioration of neurotic behaviour; (3) moderate motor restlessness; (4) variable sexual drive including a phase of hypererotism; (5) markedly altered interactions with other cats and humans; (6) slowed re-learning due to low motivation and erratic or perseverative performance on both simple and complex tasks; and (7) severe disorganization of adaptive behaviour after short periods of isolation and inactivity. The adult neurotic animals were; (8) markedly amenable to therapy although the kittens' very low resistance to the re-induction of neuroses were only mildly so; whereas (9) all showed neuroses.

In comparison, the monkeys showed (1) an initial increase in oral activity; (2) extensive amnesia for previous learning, coupled with (3) limited diminution of neurotic symptoms in the young animals as contrasted with almost complete amelioration in the adults; (4) the ability to regain pre-operative performance levels on learned problems, although the re-training period was lengthened by altered motivation, restlessness in the testing apparatus, and precipitate or inflexible responses; (5) tolerance of the proximity of or handling by a single human, contrasted with (6) fear responses to other animals or when more than one human was present; (7) increased inter-animal aggression, possibly related to (8) hyper-sexuality; and (9) diminished resistance to the re-induction of neuroses.

The theoretical implications of these findings were briefly considered.

(Authors' Abstr.)

AUGUST

Ictal Affect. *Daly, D.* 97
Commitment Procedures and the Advancement of Psychiatric Knowledge. *Gottlieb, J., and Tourney, G.* 109
A Study in Non-Restraint. *Jacoby, M. G., et al.* 114
Temporary Insanity as a Defense. *Orenstein, L. L., and Weisstein, A.* 121
The Verdict of the Kinsey Imports. *Bowman, K. M.* 126
Psychiatric Evaluation of "Normal Control" Volunteers. *Pollin, W., and Perlin, S.* .. 129
Neurotic Interaction and Patterns of Pseudo-Heredity in the Family. *Ehrenwald, J.* 134
Some Antecedent Factors in Army Prisoners. *Haas, A., and Kuras, E. J.* 143
A Singular Distortion of Temporal Orientation. *Dahl, M.* 146
The Teaching of Forensic Psychiatry in American and Canadian Medical Centers. *Stoller, R. J.* 150
Fatalities in Patients Receiving Chlorpromazine and Reserpine During 1956-1957 at Pilgrim State Hospital. *Zlotlow, M., and Paganini, A. E.* 154
*Mental Symptoms in Cases of Tumour of Temporal Lobe. *Gal, P.* 157

Mental Symptoms in Cases of Tumour of Temporal Lobe

In the series of 61 temporal lobe tumors there were mental symptoms in 50 cases. As initial symptoms there were mental manifestations in 37 cases before any neurological signs appeared. The symptoms were as follows: (1) fits (hallucinations, visual illusions, dreamy state, automatism; psychomotor equivalent, temporal epilepsy), (2) changes in personality, (3) disorders of mood, (4) schizoform psychosis, (5) parietal mental symptoms, (6) psycho-organic syndrome.

A definite mental syndrome of tumor of temporal lobe is not assumed. The author would only emphasize, that the psychic syndromes are never manifestations only of damage of a circumscribed part of the brain, but always as a damage of the function of the brain as a whole. From the clinical symptoms and their connections with the local lesions, one can only conclude that certain neuron groups play a greater part in some or in specific brain activities. On the basis of literary data, of the author's material and observations with electric stimulations of the cortex by Penfield and co-workers, it is assumed that some type of hallucinations, *déjà vu* experiences, dreamy states, automatism, psychomotor equivalence, temporal epilepsy can be—with great probability—significant of temporal lobe lesions. Paying more attention to these symptoms and making greater use of the results of other examinations it will be possible to make an earlier and more accurate diagnosis to establish the temporal lobe tumor.

(Author's Abstr.)

SEPTEMBER

Psychiatry in Asia and the Middle East. *Laughlin, H. P.* 193
Brain Mechanisms and Psychotherapy. *Eiduson, B. T.* 203
Medical, Psychiatric, and Legal Aspects of Premenstrual Tension. *Perr, I. N.* 211
The Methodological Importance of Distinguishing Two Separate Casual Chains Which Together Produce the Clinical Picture of Psychosis. *Ehrentheil, O. F.* 220
Psychiatric Orthoëpy. *Weiss, J. M. A.* 226
Toward the Effective Use of the Psychological Consultation. *Harlow, R. G., and Salzman, L. F.* 228
The Evaluation and Testing of Psychopharmaceutic Drugs. *Leveton, A. F.* 232
*Studies on Mescaline, VIII: Psychodynamic Observations. *Denber, H. C. B.* 239

Studies on Mescaline, VIII: Psychodynamic Observations

Psychodynamic observations made during the mescaline-induced state in 124 patients are reviewed. Mescaline produces a "state of being" in which the clinical spectrum can range from sleep to rage and from normalcy to thoroughly disorganized mental states. Anxiety is considered the background upon which the mescaline experience evolves. One may consider the mescaline-induced state under three headings: (1) hostility-aggression, (2) sexuality and (3) the transference. Various recurring symbolic gestures were noted, and many were frequently of a sexual nature.

The primary role of anxiety is reviewed and some theoretical concepts presented. Much further work remains to be done.

(Author's Abstr.)

OCTOBER

Research in State Mental Hospitals. *Hoch, P. H.* 289
Appraisal of the "Tranquilizers" and Their Influence on Other Somatic Treatments in Psychiatry. *Kalinowskv, L. B.* 294
*Psycho-Physiologic Evaluation of Phenyltoloxamine, A New Phrenotropic Agent: A Comparative Study with Reserpine and Placebo. *DiMascio, A., et al.* 301
Psychiatric Occupational Therapy: Some Aspects of Roles and Functions. *Ellis, M., and Bachrach, A. J.* 318
Action of Methyl-Phenidylacetate (Ritalin) and Reserpine in Behavior Disorders in Children and Adults. *Zimmerman, F. T., and Burgemeister, B. B.* 323
Psychiatric Aspects of Training for Honorable Survival as a Prisoner of War. *West, L. J.* 329
*Atropine-like Poisoning Due to Tranquilizing Agents. *Mahrer, P. R., et al.* 337
Mental Symptoms as Phenomena of Agnosia, Apraxia and Aphasia. *Fleiss, A. N.* 340
Seasonal Variation in the Incidence of Severely Crippling Mental Disorders. *Bradley, N. J., and Lucero, R. J.* 343
Delayed Complications of E.C.T.—"Crush Syndrome": Report of a Fatality. *McNichol, R.* 346
Multiple Self Concepts as Effected by Mood States. *Orzeck, A. Z., et al.* 349
Regressive EST, Chlorpromazine, and Group Therapy in Treatment of Hospitalized Chronic Schizophrenics. *King, P. D.* 354

Psycho-Physiologic Evaluation of Phenyltoloxamine, A New Phrenotropic Agent: A Comparative Study with Reserpine and Placebo

Phenyltoloxamine, a new phrenotropic agent with limited clinical experience in psychiatry, was evaluated by combined psychiatric, physiologic, and psychometric methods. A double-blind method was used comparing the drug with reserpine and placebo.

The results showed that phenyltoloxamine, at a dose of 400 mg., produced sedative and moderate hypnotic effect, reaching a peak in 4–5 hours. At peak of action it produced drowsiness and slowing of psychomotor and mental performance. This was followed by a state of relaxation, increased learning, and improved performance.

In contrast to reserpine, autonomic and visceral side effects were not observed. Muscle tension was reduced.

The findings indicated a cortical rather than sub-cortical site of action.

Its clinical uses are indicated.

The importance of multidimensional evaluation and proper methodologic control was discussed.

(Authors' Abstr.)

Atropine-Like Poisoning Due to Tranquilizing Agents

A case of atropine-like poisoning manifested by absence of sweating, hyperthermia, pupillary paralysis, dry mucous membranes, and delirium, is reported. These findings are ascribed to the anti-cholinergic properties of the tranquilizing agents of the phenothiazine group of drugs.

(Authors' Abstr.)

VOL. 12 **AM. J. PSYCHOTHER.** APRIL, 1958
Modern Psychophysiology and the Law of Initial Value. *Wilder, J.* 199
Dreams and Arousal. *Ullman, M.* 222
Drugs and Psychotherapy and the "Vital" Transference. *de Barahona Fernandes, H. J.* 243
Communication in Psychotherapy. *Barbara, D. A.* 253
Acting and Acting Out. *Bromberg, W.* 264
Re-education and Treatment of Juvenile Delinquents. *Papanek, E.* 269
Sincerity. *Schmideberg, M.* 297
Irrational Psychotherapy: An Appeal to Unreason (I). *Wolf, A., and Schwartz, E. K.* 300

JULY
The Next Ten Years in Psychiatry. *Alexander, F.* 438
Conflicting Theoretical Concepts and Practical Office Therapy. *Bronner, A.* 443
Some Variations in the Approach to Schizophrenia. *Rosen, J. D.* 451
Comments on Techniques of Termination and Transfer in a Clinic Setting. *Pumpian-Mindlin, E.* 455
Aims and Limitations of Psychotherapy. *Gruenthal, M.* 465
Language in Schizophrenia. *Brady, J. P.* 473
A Supervisor Supervised. *Markowitz, M.* 488
Training in Group Psychotherapy: A Symposium. *Beukenkamp, C., et al.* 493
Irrational Psychotherapy (II). *Wolf, A., and Schwartz, E. K.* 508

VOL. 115 **ANN. MÉD.-PSYCHOL.** 1957
Bleuler's Theories. *Minkowski, E.* 833
Should Sleep Treatment be Preserved in Psychiatry? *Monnerot, E., et al.* 845
The Foundations of a Psycho-biology. *Laborit, H.* 881
Neuropsychiatric Accidents in Tubercular Patients Treated with Cycloserine. *Lévi-Valensi, et al.* 899

VOL. 116 1958
Why I do not Think Much of Modern Schizophrenia. *Abely, P.* 1
Some Indications which Delimit the Different Phases of Human Activity, etc. *Rezal, M.* 44
The Genesis of Antisocial Behaviour in the Hospitalised Mental Patient. *Vallet, R.* 48
General Paralysis in Africa. *Rainaut, J.* 209
Factors of Cohesion in Social Behaviour in a Psychiatric Hospital. *Fernandez, A., et al.* 222
Infantile Neuroses Showing Themselves by Somatic Symptoms. *Jakab, I.* 252
The Chemical Study of the Constituents of Cerebral Tissue During the Nineteenth Century. *Chatagnon, C., and P.* 267
Psychosociological Researches on Mental Illness of a Free Service. *Delay, J., et al.* 417
Assistance to Backward Children in Belgium. *Beley, A.* 441
Neuropsychiatric Complications and EEG Modifications during Treatment of Pulmonary Tuberculosis by Cycloserine. *Naquet, N., et al.* 462
The Neuropsychiatric Syndrome in Sparmophilia. *Joyeux, J.* 473
Congenital Indifference to Pain with Analgesia and Agnosia with Confinement. *Gayral, L.* 593
Endocrine Disorders in the Phacomatoses. *Alliez, J., and Montin, P.* 603
The World of Prometheus Unbound. *Loras, O.* 624
Infantile Schizophrenia. *Reauyard, M.* 785
Notes on the Psychology of Stabilized and Ameliorated Schizophrenia. *Fouks, L., et al.* 805
The Modern Conception of Schizophrenia in the Light of the Rorschach Test, etc. *Copelman, L. S.* 815
Results Obtained by the Treatment of Chronic Alcoholism with Vitamins in Big Doses. *Boittelle, G., et al.* 823
Late Results of Lobotomy. *Descuns, P., et al.* 855

VOL. 117
Some Signs of Apraxia in Children. *Wallon, H., and Denjean, G.* 1
Paraphrenia. *Durand, V. J., et al.* 15
The Interpretation of the Central Action of Certain Psychoactive Reagents (Serotonine in particular), etc. *Laborit, H., et al.* 60
Comparative Activity of Certain Neuroleptic Drugs (hydroxyzine, miltown, acetopromazine and 7044R.P.). *Pnech, J., et al.* 79
Erotomania of Clerambault, a Proud Erotism. *Leconte, M.* 209
Social Levels and Interindividual Behaviour in a Psychiatric Milieu. *Fernandez-Zoila, A., and Lebretou, M.* 224
Psychopathological Repercussions of Political Conflicts of Algerians, etc. *Valladi, L.* 255
Recurrent Fever in the Treatment of G.P.I. *Mareschal, P., and Noblet, J.* 268
A Rare Case of Criminal Sexual Inversion. *Popescu-Sibiu, I.* 279
Fractional Encephalography and the EEG in the Manic-Depressive Psychosis. *Borenstein, P., et al.* 417
Heterosexual Heantoscopy in Schizophrenia. *Letailleur, M., et al.* 451

The "Economisers of Insulin" in Sakel's Treatment (Hyaluronidase and BZ55). *Racarnier, P.-C., et al.* 462
The Psychogenesis of Alcoholism and the Psychotherapeutic Attitude. *Carrere, J.* 481
The Treatment of Acute Alcoholic Syndromes with Viadril. *Ducoudray, J., and Noyer, J.* 496

VOL. 2 ARCH. CRIM. PSYCHODYN. 1957

The Murder and the Murderer, the Destroyer and the Creator. *Bromberg, W.* 523
"The Songs of Bilitis", A Voyage in Lesbianism. *Winzie, G. B., Jr.* 530
Uses of Delinquent Behavior by a Borderline Schizophrenic. *Boyer, L. B.* 541
The Configurational Approach to Causation in the Study of Juvenile Delinquents. *Frankenstein, C.* 572
Dream Life in a Case of Uxoricide (Part I). *Karpman, B.* 597

VOL. 79 ARCH. NEUR. PSYCHIAT. APRIL, 1958

The Electroencephalogram in Diffuse Encephalopathies. *Lesse, S., et al.* 359
Experimental Study of Distant Effects of Acute Focal Brain Injury. *Kempinsky, W. H.* 376
Relationship Between Anticoagulants and Hemorrhagic Cerebral Infarction in Experimental Animals. *Wood, M. W., et al.* 390
Classification and Rating of Emotional Experiences. *Hamburg, D. A., et al.* 415
Relation Between Fine and Gross Psychomotor Movement in Schizophrenia. *King, H. E.* 427
*Relation of Emotional Responses and Changes in Plasma Hydrocortisone Level After Stressful Interview. *Persky, H., et al.* 434
Psychiatrists' Conceptions of the Schizophrenogenic Parent. *Jackson, D. D., et al.* 448
Effectiveness of Insulin Coma in the Treatment of Schizophrenia. *Patterson, E. S.* 460
Chlorpromazine and Communication Processes. *Tourlentes, T. T., et al.* 468

Relation of Emotional Responses and Changes in Plasma Hydrocortisone Level after Stressful Interview

The present report has attempted to investigate the relationship between emotional response and the plasma level of hydrocortisone under the impact of a stressful interview. Two major areas of psychological-endocrine interaction were examined: (1) the effect of emotional response and (2) the effect of the psychological stimulus on the change in the hormone level. The degrees of increase in anxiety, anger, depression, and a combined affect rating were found to be significantly and linearly related to the change in the plasma hydrocortisone level. This relationship was maintained even though the affect change was rated for only a short time period. It would appear that the plasma level of hydrocortisone is increased by any type of emotional arousal. However, data were presented which indicated a particularly striking effect when anxiety of a disintegrative nature was developed in the course of the stress interview. Stimulus-hormone relationships were also found to occur, but these relations were generally less striking than response-hormone relations. The stresser's attitude and his total stimulus intensity rating tended to parallel the change in plasma hormone level. A significant stimulus-hormone relationship occurred when the stimulus produced a relatively intense emotional response in the subject. The findings presented are of special interest because this experiment included only a moderate range of emotional responses. The limited data available on more extreme responses suggest that an even greater degree of adrenocortical activation occurs.

(Authors' Abstr.)

MAY

*Memory Deficit Produced by Bilateral Lesions in the Hippocampal Zone. *Penfield, W., and Milner, B.* 475
Encephalography by the Displacement Technique. *Nelson, D. A., et al.* 498
Loss of Consciousness and Convulsions with Congenital Heart Disease. *Tyler, H. R., and Clark, D. B.* 506
*Effect of Hypothalamic Lesions on the Amygdala Syndrome in the Cat. *Kling, A., and Hutt, P. J.* 511
Interrelations of Mesial Temporal and Orbital Frontal Areas of Man Revealed by Strychnine Spikes. *Kendrick, J. F., and Gibbs, F. A.* 518
Psychiatric Problems of a Rapidly Growing Suburb. *Gordon, R. E., and K. K.* 543
Autonomic and Adrenal Responsivity in Psychiatric Patients. *Sloane, R. B., et al.* 549
Patient and Therapist Observations on the Circumstances of a Schizophrenic Episode. *Weakland, J. H., and Jackson, D. D.* 554
Ascorbic Acid Level and Lag Time in Oxidation of N,N-Dimethyl-*p*-Phenylenediamine. *Aprison, M. H., and Grosz, H. J.* 575
*Studies in the Effect of Lysergic Acid Diethylamide (LSD-25). *Liebert, R. S., et al.* 580
Reliability of the Methacholine (Mecholyl) Test. *Maas, J. W.* 585
Effects of a Tranquilizer (Reserpine) on Psychodynamic and Social Processes. *Savage, C., and Day, J.* 590
Chemotherapeutic Trials in Psychosis. *Turner, W. J., et al.* 597
Studies in Psychophysiology of Dreams. *Wolpert, E. A., and Trosman, H.* 603

Memory Deficit Produced by Bilateral Lesions in the Hippocampal Zone

Bilateral removal of the hippocampus and hippocampal gyrus in man produces loss of recent memory. As soon as he has turned his attention to something else, the patient is unable to remember what was happening a moment earlier. It is as though he had made no record of present experience. He also has a retrograde memory loss that covers a considerable period before operation.

This is a confirmation of the suggestion of Glees and Griffith, based on the evidence of autopsy study of a patient with bilateral brain lesions. "The suggestion is made," they wrote, "that the hippocampal formation of the adult seems to be essential for recent memory." In their case, as well as in our cases, the hippocampal gyrus was destroyed along with the hippocampus.

Psychological study of our patients shows that, in spite of the above-noted deficiencies, memory for the distant past is not lost, nor is there corresponding loss of attention, concentration, reasoning ability, or previously acquired skills. The intelligence quotient shows no drop when compared with pre-operative testing. There is no interference with speech, and the memory of words is unimpaired.

It may be concluded that an essential part of the recording mechanism is contained in the hippocampal zone. Certainly, also, another part of the mechanism lies in the central integrating circuits of the brain stem, circuits that have duplicate connections with the two hippocampal areas.

These conclusions are based largely on the post-operative study of four patients in whom this distressing complication appeared. Two of our own were found to have this defect after unilateral excisions had been carried out in the treatment of temporal-lobe epilepsy, the contralateral hippocampal zone apparently having been injured by incisural herniation at birth. Two patients were added, for us to study, in whom bilateral hippocampectomy had been carried out by Dr. William Scoville, in his search for a better operation to replace frontal lobotomy as a treatment for hopeless psychotics. The symptom complex was identical in all four.

This case analysis was undertaken originally because of remorse that two of our patients should have suffered a complication. It is a severe handicap to them, although both continue to earn their living, which had been threatened by the frequency of their pre-operative cerebral seizures. But the study, which was intended primarily to serve as a warning so that others, as well as ourselves, might avoid this pitfall, has opened up new and exciting evidence.

Neurosurgeons who hope to cure temporal-lobe seizures by removal of one anterior temporal lobe and all of the hippocampal zone should bear in mind that the squeezing of the baby's head at the time of birth, which produces temporal herniation and incisural sclerosis, may occasionally destroy the hippocampal zone on the other side. Thus the major epileptogenic discharge may be on the side of the less severe injury. Removal of the zone on that side might then be expected to result in this loss of recent memory. Such a loss is what occurred in the two cases that have been reported.

A careful psychological study of more than 90 other patients was carried out before and after the same unilateral operation, and they showed no evidence of general memory loss. Under normal conditions one hippocampal area duplicates the work of the other and carries on when one is removed.

For the time being, in order to guard against such memory losses, the authors have adopted the policy that when the initial electroencephalographic study points to independent abnormality in both inferior temporal regions, no removal should be made of either hippocampal zone.

The analysis of this complication of recent memory loss, undertaken as a neurosurgical project, has thrown welcome light on the location of some portion at least of the ganglionic record of current experience. It has demonstrated that this record is anatomically separate from the neurological mechanisms that are related to words, skills, and concepts that have been preciously acquired.

(Authors' Abstr.)

Effect of Hypothalamic Lesions on the Amygdala Syndrome in the Cat

In the cat the "amygdala syndrome" does not appear after bilateral ablation of the amygdaloid complex if there has been previous destruction of the ventromedial hypothalamic nuclei or the mammillary bodies; and the amygdala syndrome, once produced by the amygdala ablation can be abolished by the hypothalamic lesions. Examples of lesions and theoretical considerations are included.

(Authors' Abstr.)

Studies in the Effect of Lysergic Acid Diethylamide (LSD-25)

Under the influence of lysergic acid diethylamide (LSD-25), for both normals and schizophrenics, there is a significant increase in the perceived size of one's own body and its parts, and no significant change in the perceived size of external objects. These findings are interpreted in terms of the assumption that LSD operates as a primitivizing agent, which is assumed to lessen the definiteness of the boundary of the body in relation to the surroundings.

(Authors' Abstr.)

JUNE

Gliomas of the Optic Nerves. *Dodge, H. W., Jr., et al.* 607
Intracranial Arteriovenous Aneurysms. *Höök, O., et al.* 622
Clinical Correlates of Electroshock Therapy. *Aird, R. B.* 633
Retention of Overlearned Visual Habit After Temporal Cortical Ablation in Monkey. *Chow, K. L., and Survis, J.* 640
Newcastle Disease Encephalomyelitis in Cats. *Luttrell, C. N., and Bang, F. B.* 647
Psychotherapy in a Home for the Aged. *Aronson, M. J.* 671
Measure of the Inadequacy in Schizophrenic Patients for Making Difficult Discriminative Evaluations. *Heath, H. A.* 675
Psychotherapy with Ambulatory Schizophrenic Patients in Mixed Analytic Groups. *Hulse, W. C.* 681
A Quantitative Method of Estimating Variations in Intensity of a Psychologic Conflict or State. *Gottschalk, L. A., and Kaplan, S.* 688
Chlorpromazine Used with an Intensive Occupational Therapy Program. *Grygier, P., and Waters, M. A.* 697
Dynamic Aspects of Occupational Therapy. *Wittkower, E. D., and Azima, H.* 706
Analeptic Action of Lysergic Acid Diethylamide (LSD-25) Against Pentobarbital. *Apter, J. T.* 711
Constancy of the Funkenstein Test. *Brill, N. Q., et al.* 716
Oculomotor and Postural Patterns in Schizophrenic Children. *Pollack, M., and Krieger, H. P.* 720
Responsivity of Adrenal Gland in Schizophrenia to Corticotropin. *Sheard, M. H.* 727
*Ceruloplasmin, Transferrin, and Tryptophan in Schizophrenia. *Frohman, C. E., et al.* 730

Ceruloplasmin, Transferrin, and Tryptophan in Schizophrenia

Thorough investigation of copper and ceruloplasmin levels in normal and schizophrenic subjects gave no indication that these substances are altered quantitatively in schizophrenia. It remains to be shown whether or not qualitative differences exist in this disease. The same study of iron and transferrin levels shows that, while schizophrenic subjects have lowered quantities of these substances, the levels in the other patients studied were lowered as much or more. Investigation of tryptophan excretion of normal and schizophrenic subjects on normal-, high-, and low-tryptophan diets gave no indication that increased tryptophan excretion was associated with schizophrenia. The findings give clear evidence that diet must be rigidly controlled before metabolic investigations can be considered to be of significance.

(Authors' Abstr.)

VOL. 80 JULY 1958

Paramyotonia Congenita. *Drager, G. A., et al.* 1
Myasthenia Gravis and Epilepsy. *Hoefer, P. F. A., et al.* 10
Tumors of the Basal Ganglia. *Arseni, C.* 18
Electrical Activity of the Hippocampus of Patients with Temporal Lobe Epilepsy. *Kajtor, F., et al.* 25
Intracranial Arteriovenous Aneurysms. *Höök, O., and Johanson, C.* 39
The Experimental Subject. *Perlin, S., et al.* 65
Feasibility of Community Clinic Treatment for State Mental Hospital Patients. *Sampson, H., et al.* 71
*Urinary Excretion of 5-Hydroxyindoleacetic Acid in Psychotic and Normal Subjects. *Buscaino, G. A., and Stefanachi, L.* 78
The Patient and His Experiences in an Outpatient Clinic. *Katz, J., and Solomon, R. Z.* 86
Hallucination as an Ego Experience. *Hollender, M. H., and Böszörményi-Nagy, I.* 93
Experience of Perceptual Distortion as a Source of Anxiety. *Korchin, S. J., et al.* 98
Emotional Determinants of Mental Deficiency. *Eisenberg, L.* 114
*Observations on Schizophrenic Patients After Iproniazid and Tryptophan. *Lauer, J. W., et al.* 122

Urinary Excretion of 5-Hydroxyindoleacetic Acid in Psychotic and Normal Subjects

The daily excretion of 5-hydroxyindoleacetic acid (5-HIAA) in schizophrenics does not differ significantly from that of non-schizophrenic subjects. If in schizophrenia there is a basic alteration of the metabolism of 5-hydroxytryptamine, it is not reflected in the amount of its endmetabolite, 5-HIAA, excreted in the urine.

Parenterally injected 5-hydroxytryptamine may appear in the urine as 5-HIAA. In non-schizophrenic subjects the recovery rate averages 25 per cent., while in schizophrenic patients it averages 42 per cent., with individual values higher than 90 per cent. in some acute schizophrenics.

Chromatographic study has revealed the presence of indole metabolites different from 5-HIAA in the urines of catatonic patients and of acute schizophrenics in general. Such metabolites are perhaps responsible for the high incidence of positive results in the qualitative colour reaction, although the urinary concentrations of this substance are not increased. The results do not support the hypothetical existence of a primary disturbance of systemic serotonin metabolism in schizophrenia. The abnormalities found in some acute schizophrenics, especially the catatonics, are regarded as added evidence for an enzymatic disorder affecting the general metabolism of biological amines in the early stages of schizophrenia.

(Authors' Abstr.)

Observations on Schizophrenic Patients After Iproniazid and Tryptophan

Seven schizophrenic patients were given iproniazid and L-tryptophan for a period of six weeks.

During the experimental period, the patients exhibited an increase in energy level and motor activity and improvements in the ability to accept interpersonal relationships, and displayed more affect. In addition, their voluntary intake of protein and tryptophan increased. These changes became apparent about two weeks after the initiation of the experimental period.

During the course of the experiment the 5-hydroxyindoleacetic acid excretion per 24 hours diminished. This low excretion was rapidly reversible after the cessation of drug administration.

In a tryptophan-load test 26 schizophrenic patients did not exhibit any increase in 5-hydroxyindoleacetic acid excretion over the basal level after tryptophan administration, while 17 controls showed an 100 per cent. increase in the excretion of this compound. No difference between the two groups at the basal level was observed.

Some implications of the results obtained in relation to serotonin metabolism are discussed.

(Authors' Abstr.)

AUGUST

Clinical Features of Porencephaly. *Naef, R. W.* 133
Cerebrospinal Fluid Transaminase and Lactic Dehydrogenase Activities in Neurologic Disease. *Green, J. B., et al.* 148
Serum Aldolase in Muscular Dystrophies, Neuromuscular Disorders, and Wasting of Skeletal Muscle. *Rowland, L. P., and Ross, G.* 157
Pelizaeus-Merzbacher Disease. *Tyler, H. R.* 162
Intracranial Hemorrhage in Cerebral Arteriovenous Anomalies. *Wood, M. W., and Svien, H. J.* 170
Autoscopic Phenomena. *Lukianowicz, N.* 199
*Cerebrospinal Fluid Neuraminic Acid Deficiency in Schizophrenia. *Bogoch, S.* .. 221
The Digit Span Test and the Prediction of Cerebral Pathology. *Heilbrun, A. B., Jr.* .. 228
The Sedation Threshold. *Kawi, A. A.* 232
Blood Glutathione Levels in the Male Schizophrenic Patient. *Barak, A. J., et al.* .. 237
Personality Differences and Continued Meprobamate and Proclorperazine Administration. *Kelly, E. L., et al.* 241
Continued Meprobamate and Proclorperazine Administration and Behavior. *Kelly, E. L., et al.* 247
Homosexuality in College. *Ross, M., and Mendelsohn, F.* 253
A Study of the Development of Olfactory Preferences. *Stein, M., et al.* 264

Cerebrospinal Fluid Neuraminic Acid Deficiency in Schizophrenia

1. The mean neuraminic acid content of cerebrospinal fluid in schizophrenic patients is shown to be considerably below that of non-schizophrenic adults, and comparable only to values found in some children under 7 years of age.

2. The method employed for the preparation of crystalline neuraminic acid from purified bovine brain ganglioside for use as standard is detailed.

3. The method employed for determination of neuraminic acid in cerebrospinal fluid, together with the precautions to be observed, is described.

4. The concept of a barrier-antibody system and its possible relationship to the psychoses is presented. The susceptibility of this hypothesis to experimental validation or negation is stressed.

5. It is concluded that an important new area for further investigation has been indicated.

(Authors' Abstr.)

SEPTEMBER

Abnormalities in Central Regulation of Respiration in Acute and Convalescent Poliomyelitis. *Plum, F., and Swanson, A. G.* 267
Neurological Syndromes Associated with Congenital Absence of the Odontoid Process. *Rowland, L. P., et al.* 286
Neurochemistry and Therapeutic Endeavor. *McIlwain, H.* 292
Interhemispheric Communication Through the Corpus Callosum. *Myers, R. E., and Sperry, R. W.* 298
*Cerebrospinal Fluid Free Cholesterol as Index of Activity of Multiple Sclerosis and Allied Diseases. *Poser, C. M., and Curran, G. L.* 304
Cortical and Rhinencephalic Electrical Potentials During Hypoglycemia. *Van Meter, W. G., et al.* 314

A Combined Biochemical and Clinical Study of Mental Disease. *Harris, W. H., and Patterson, E. S.* .. 335
Lysergic Acid Diethylamide (LSD-25). *Weiss, B., et al.* .. 345
Psychoanalytic Orientation in Psychotherapy. *Lehrman, S. R.* .. 351
Development of an Active Therapeutic Program on a Chronic Ward. *Wadeson, R. W., Jr., et al.* .. 363
Mechanisms Involved in the Cardiovascular Response to Transcranial Stimulation. *Colville, K. I., et al.* .. 374
Effect of Anticholinergic Agent, Diethazine, on EEG and Behavior. *Fink, M.* .. 380
*Azacyclonol (Frenquel) in Chronic Schizophrenia. *Hollander, L., et al.* .. 388

Cerebrospinal Fluid Free Cholesterol as Index of Activity of Multiple Sclerosis and Allied Diseases

Free and esterified cholesterol was determined in the CSF, using a turbidimetric method for the estimation of digitonin-precipitable sterols.

Esterified cholesterol, but no free cholesterol, was found in the spinal fluid of normal controls.

In infections and neoplasms of the nervous system, the elevation of total- and free-cholesterol fractions was found to correlate with the elevation of the spinal fluid protein.

In amyotrophic lateral sclerosis, multiple sclerosis, and other similar demyelinizing diseases, the CSF cholesterol is independent of the level of CSF protein. The correlation between free CSF cholesterol and the clinical evaluation of activity of the disease would seem to indicate that the presence of free cholesterol may be useful as an objective index of activity of the demyelinizing process.

(Authors' Abstr.)

Azacyclonol (Frenquel) in Chronic Schizophrenia

Nineteen chronic schizophrenic men, divided randomly into two groups, were intensively studied by a double-blind crossover design for a total of five months to evaluate the effectiveness of azacyclonol (Frenquel). A daily dose of 120 mg. of azacyclonol had no significant effect on the thinking or behaviour of these chronic schizophrenic patients. Extravagant claims for this drug made by some previous investigators were not supported by this study.

(Authors' Abstr.)

OCTOBER

Specific and General Effects of Brain Injury in Man. *Teuber, H.-L., and Liebert, R. S.* 403
Recall with Amobarbital (Amytal) Sodium in Diagnosis of Seizures. *Collings, H., Jr.* 408
Withdrawal Convulsions in Dogs Following Chronic Meprobamate Intoxication. *Essig, C. F.* .. 414
Cinephotomicrography of the Pial Circulation. *Gurdjian, E. S., et al.* .. 418
Lumbar Extradural Cysts—Congenital. *Smith, G. W., and Chavez, M.* .. 436
*Effect of Blood Plasma from Psychotic Patients upon Performance of Trained Rats. *Winter, C. A., and Flataker, L.* .. 441
Depersonalization. *Bird, B.* .. 467
*Effects of Lysergic Acid Diethylamide (LSD-25) on Intellectual Functions. *Silverstein, A. B., and Klee, G. D.* .. 477
*Thiopropazate Hydrochloride (Dartal) Chemotherapy for Emotional Disorders. *Edisen, C. B., and Samuels, A. S.* .. 481
The Stability of Epinephrine and Arterenol (Norepinephrine) in Plasma and Serum. *Cohen, G., et al.* .. 484
Current Status of the Funkenstein Text. *Feinberg, I.* .. 488
Ambiguity and Repression. *Kepecs, J. G.* .. 502
The Organization Factor as an Explanatory Principle in Functional Psychosis. *Rashkis, H. A.* .. 513
Explorations in Psychotherapy. *Bockoven, J. S.* .. 520

Effect of Blood Plasma from Psychotic Patients upon Performance of Trained Rats

Intraperitoneal injection of blood plasma from the majority of schizophrenic and other severely psychotic patients into rats produced a syndrome resembling, but not identical with, that produced by injection of LSD. In rats trained to climb a rope, the injection of 1 ml. of plasma produced a deficiency in performance which could be objectively and quantitatively measured. Comparisons of the effects of plasma from 80 psychotic patients and 82 non-psychotic subjects, including both general hospital patients and normal subjects, showed a marked and highly significant difference between the two groups. Cerebrospinal fluid had much less effect than plasma.

(Authors' Abstr.)

Effects of Lysergic Acid Diethylamide (LSD-25) on Intellectual Functions
I. Memory

LSD-25 in doses of 72 μg. significantly impaired memory for only certain kinds of material, as measured by the Wechsler Memory Scale. On the one hand, the ability to draw geometric figures from memory, the ability to reproduce brief prose passages, and the ability to count backward, say the alphabet, and perform serial addition were all affected by the drug. On the other hand, the ability to learn paired associates, the ability to repeat series of digits, orientation for time and place, and memory for personal and current information appeared to be unaffected by the drug at the dose level employed.

II. Abstract Thinking

LSD-25 in doses of 72 μg. significantly impaired abstract thinking, as measured by the Gorham Proverbs Test. The difference in scores under control and under drug conditions resembled the difference reported in the scores of normal subjects and chronic schizophrenics. Contrary to expectations incorrect abstractions were commoner than concrete responses not only under control conditions, but also in the drug state.

(Authors' Abstr.)

Thiopropazate Hydrochloride (Dartal) Chemotherapy for Emotional Disorders

Thiopropazate hydrochloride (Dartal) chemotherapy has been employed in the treatment of 104 psychiatric patients, the majority of whom were schizophrenics. The drug has a definite ataractic effect for both acute and chronic disturbances, and 77 patients (74 per cent. of the total group) showed some overall improvement on the drug. The drug is particularly useful in treating patients with association defect, depersonalization, and anxiety; and, although the statistical significance of the drug effect is not as definitive, it also appears to be helpful in treating patients manifesting delusions, withdrawn sociability, flattened affect, and anorexia. Patients with mood depression do not respond to thiopropazate therapy and demonstrate greater improvement when taking placebo.

No serious side-effects were encountered, but the development of excitation and Parkinsonism-like symptoms in some patients produces considerable secondary anxiety, necessitating discontinuation of the medicine. No evidence of myeloid depression has been found. On a milligram basis, thiopropazate hydrochloride is about five times as potent as chlorpromazine (Thorazine), and the recommended dosage is 5 to 10 mg. four times a day.

(Authors' Abstr.)

VOL. 81 **BRAIN** JUNE, 1958

Recurrent Polyneuropathies and their Corticosteroid Treatment. *Austin, J. H.*	157
Tonic Seizures in Disseminated Sclerosis. *Matthews, W.*	193
The Blood Vessels of the Human Optic Chiasma and their Relation to those of the Hypophysis and Hypothalamus. *Dawson, B. H.*	207
The Groping Phenomena of the Foot. *Fradis, A., and Botez, M. I.*	218
Centrencephalic Integrating System. *Penfield, W.*	231
Internal Hydrocephalus Caused by Parasitic Cysts. *Kuper, S., et al.*	235
The Spongy Type of Diffuse Sclerosis. *Wolman, M.*	243
The Blood-Brain Barrier: An Experimental Study with Vital Dyes. *Millen, J. W., and Hess, A.*	248
The Diagnosis and Treatment of Wilson's Disease. *Warnock, C. G., and Neill, D. W.*	258

VOL. 49 **BR. J. PSYCHOL.** MAY, 1958

Partial Advance Information in a Choice Reaction Task. *Leonard, J. A.*	89
Factors Affecting Inductive Predictions. *Green, R. T.*	97
Experimental Design: A Cloak for Intellectual Sterility. *Taylor, J. G.*	106
The Effect of Set on Accuracy of Auditory Perception. *Talland, G. A.*	117
Tachistoscopic Exposure Time and Spatial Proximity in the Organization of Visual Perception. *Kaswan, J. W.*	131
Psychomotor Control in Stammering and Normal Subjects: An Experimental Study. *Seth, G.*	139
Cognitive and Social Changes in the Feebleminded—Three Further Studies. *Clarke, A. D. B., et al.*	144
Shape Recognition: A Reply to Deutsch. *Dodwell, P. C.*	158

AUGUST

Jones, Ernest: Obituary Notice	177
Visually Controlled Locomotion and Visual Orientation in Animals. *Gibson, J. J.*	182
Experiments on "Neophobia" in Wild and Laboratory Rats. *Barnett, S. A.*	195
The Role of the Reinforcer as a Stimulus. *Reid, R. L.*	202
Visual Search for Successive Decisions. *Mackworth, N. H., and J. F.*	210
The Role of Attitudes in Comprehension. *Friedmann, S.*	222
Time for Reading and Memory. *Poulton, E. C.*	230
Effects of Practice and Rest on Fluctuations in the Müller-Lyer Illusion. *Eysenck, H. J., and Slater, P.*	246

VOL. 23 **BULL. LOS ANGELES NEUR. SOC.** MARCH, 1958

Value of the Electromyogram in Carpal Tunnel Syndrome. *Marinacci, A. A.*	1
Mental Deficiency from Intrauterine Exposure to Radiation. *Courville, C. B., and Edmondson, H. A.*	11

Spinal Epidural Abscess. Review of the Literature and Report of Case. *Korbin, W. M.* 21
Hereditary Olivopontocerebellar Atrophy. *Nielsen, J. M., and Hyman, W.* 27
Vascular Patterns of the Encephalic Gray Matter in Man. *Courville, C. B.* 30
Intracranial Epidermoid Tumor in an Eighty-Year-Old Man. Report of Case. *Broussalian, S. L.* 44

JUNE

Syndrome of Muscular Fasciculations and Atrophy. *Nielsen, J. M., and Marvin, S. L.* 51
The Use of Electromyography in the Differential Diagnosis of Lumbar Herniated Disks. *Marinacci, A. A.* 65
Gangliocytoma Myelinicum Diffusum of the Cerebellar Cortex. Review of the Literature and Report of Case. *Courville, C. B.* 72
Surgical Approaches to Craniopharyngeal Tumors. Total Resection with Adjunctive Hormonal Therapy. *Vogel, P. J.* 81
Calcification and Ossification Incident to Compressive Softening of the Spinal Cord. Report of Case. *Courville, C. B.* 88
Case of Alexia and Visual Agnosia for Objects. *Boyle, D. G., and McKeown, M.* .. 92

SEPTEMBER

Telangiectasis of Skin and Spinal Cord. *Nielsen, J. M., et al.* 97
Infectious Neuronitis and Its Electromyographic Characteristics. *Marinacci, A. A.* .. 102
Anterolateral Cordotomy for Intractable Pain Following Unsuccessful Disc Surgery. A Review of Fourteen Cases. *Abbott, K. H., and Retter, R. H.* 112
Intracranial Ependymochoroidal Tumors. An Analysis of Fifty-three Cases. *Broussalian, S. L.* 119
Clinical Experience with Peganone. A Preliminary Report. *Mann, L. B.* 130
Changes in the Vasculature of the Encephalic Gray Matter in Marked Congestion (Polycythemia Vera). *Courville, C. B.* 134

VOL. 34 CERVELLO JANUARY, 1958

A Case of Pick's Disease with Temporal Localisation and with Unilateral Lesion in the Optic Thalamus. *Fattovich, G.* 1
Serum and Urinary Mucoids in Recent and Chronic Schizophrenia. *Papalia, A., and Scarcella, M.* 23
Unilateral Gynaecomastia Associated with a Schizophrenic Syndrome. *Pellagrini, G., and Serini, G.* 42
Hypoglycemic Treatment with Insulin and BZ55 in Mental Diseases. *Pelizzari, G., and Filippini, V.* 55

MARCH

A Case of Megalencephalia. *Fattovich, G.* 81
Antistreptolysin in Neurology. *Sassi, A.* 99

MAY

A Case of Pick's Disease with Extremely Rapid Course. *Fattovich, G.* 153
Considerations of Some Cases of Craniocerebral Trauma in Relation to Traumatic Epilepsy. *Simonetti, L.* 171

JULY

Importance of Haematology in Mental Pathology. *Del Bo Rossi, L.* 233
Statistical Importance and Therapeutic Considerations on Recovery from Delirium Tremens, etc. *Pellegrini, G., and Serini, G.* 239
The Anti-convulsant Effects of Meprobamate. *Maria, G.* 253

SEPTEMBER

On the Problem of Mental Syndromes in Hyperthyroidism and After Thyroidectomy. *Bonetti, U.* 305
A Common Factor in Our Treatment of Mental Pathology. *Del Bo Rossi, L.* .. 339
The Pathology of Demyelinating Diseases. *Peters, G., et al.* 343
The Cytology of the Neurone. *Seitelberger, F., et al.* 363

VOL. 17 CONF. NEUR. 1957

The Subependymal Zone of the Human Cerebellar Nodulus. *Rae, A. S. L.* 253
Tuberculomata of the Central Nervous System. Brain and Spinal Cord. *Arseni, C.* .. 258
Studies of the Proteins of Human Cerebrospinal Fluid by Paper Electrophoresis. *de Risio, C., et al.* 271
Studies in Stereoencephalotomy: VII. Variations in the Position of the Globus Pallidus. *Baird, H. W., III, et al.* 288
Studies in Stereoencephalotomy: VIII. Comparison of the Variability of Subcortical Lesions Produced by Various Procedures (Radio-frequency Coagulation, Electrolysis, Alcohol Injection). *Gildenberg, P. L.* 299

Lipases in the Cerebrospinal Fluid in Various Neurological Conditions, Especially Infantile Amaurotic Idiocy (Tay-Sachs Disease). *Spiegel-Adolf, M., et al.* .. 310
Subacute Necrotic Myelopathy with Bulbar Involvement. *Behar, A. J., et al.* .. 329
Tetaniform Convulsions Precipitated by Fluoridated Drinking Water. *Waldbott, G. L.* 339
Suprasegmental Lesions in Friedreich's Ataxia. *Urich, H., et al.* 360
The Occurrence of Erb's Palsy in Three Siblings of One Family. *Adler, E., and Eliakim, C.* 371

VOL. 18 1958
Giant-Cell Tumor of the Spine. *Weinberg, M.-H.* 1
Disseminated Encephalitis Beginning with Symptoms of Lactation Neuritis of the Optic Nerve. *Kepes, J., and Remenár, L.* 12
Pathophysiological Aspects of Psychoses. Symposium at the IInd International Congress of Psychiatry, Zurich, 1–7 September, 1957 69–320
Parkinsonism with Oculogyric Crises. Stimulation and Partial Elimination of Periaqueductal Grey and Mesencephalic Tegmentum (Tegmentotomy). *Wycis, H. T., and Spiegel, E. A.* 385
A Contribution to the Problem of Extracranial Metastasis in Meningiomas. *Baumann, J.* 394
Some Laboratory Investigations in Multiple Sclerosis. *Jeanes, A. L., and Cumings, J. N.* 397
Thrombocyte Agglutination in Disseminated Sclerosis and Other Neurological Diseases. *Persson, I.* 405
Personality Changes Following Thalamic and Pallidal Lesions Compared with the Effects of Various Cortical Lesions. *Orchinik, C. W., and Petrie, A.* 442

VOL. 19 **DIS. NERV. SYST.** JULY, 1958
The Basis of Effective Military Psychiatry. *Bell, E., Jr.* 283
Factors Affecting Results in Psychotherapy. *Oremland, J. D., et al.* 289
Intravenous Thiobarbiturates and Electroshock Therapy. *Kerman, E. F.* 295
Hexobarbital Sodium in Clinical EEG. *Merillat, H. C., et al.* 297
Correlation of Glucose Insulin Tolerance Test with Insulin Coma Therapy. *Read, M. R., and Kaufman, J. S.* 299
Cigarette Neurotics. *Bergler, E.* 305
Effect of Intravenous Promazine on Arousal Responses in Man. *Wilson, W. P., and Glotfelty, J. S.* 307
High Dosage Compazine in Chronic Schizophrenia. *Holman, W. T.* 309

JULY
MONOGRAPH SUPPLEMENT
An Appraisal of Psychogenetic Twin Data. *Kallmann, F. J.* 9
Recent Developments in Behavior Genetics and Differential Psychology. *Hirsch, J.* .. 17
Hereditary Defects in Protein Synthesis as Related to Psychiatry. *Scheinberg, I. H.* .. 25
Patterns of Discovery in the Genetics of Mental Deficiency. *Allen, G.* 31
Function of the Anesthetist in Electroshock Therapy. *Alexander, L., et al.* 41
Newer Psychiatric Drugs. *Holt, W. L., Jr., et al.* 47
Experimental Investigations. *Gahagan, L. H., et al.* 54
Value of Non-Convulsive Technics. *Campden-Main, B. C., et al.* 64
Technic of Complete Myoneural Blockade in Psychiatric Electrotherapy. *Gahagan, L. H., and Richardson, D. J.* 71
Succinylcholine Chloride and Thiopental Preparatory to Electroconvulsive Therapy. *Gilbert, I. E., et al.* 72
Greater Safety by Softer Electroconvulsive Treatment with SCC and Neraval. *Robie, T. R.* 73
PM-GM Succinylcholine Modified Electroshock Therapy. *Gabriel, A. R.* 75
PM-GM Succinylcholine (SCC) Technic. *Erdos, J. M.* 76
Succinylcholine Modified Electroconvulsive Treatment. *Holt, W. L., Jr.* 77
Electric Stimulation of the Diencephalon. *Breitner, C.* 80
Multipolar Electrocoma Therapy. *Friedman, E.* 83
Electro-Sleep in a Chronic Disturbed Service. *Fenichel, M.* 84
Abreaction Produced by Low Non-Convulsive Electrostimulation. *Wilcox, P. H.* .. 87

AUGUST
Establishing a New Psychiatric Department Within a Medical School. *Caveny, E. L., and Sussex, J. N.* 327
The Psychiatrist's Responsibilities. *Liebman, S., et al.* 332
Thyroid Function in Mental Diseases. *Stevens, J. D., and Dunn, A. L.* 338
Results with Chlorpromazine on Chronically Nude, Incontinent, Combative and Noisy Patients. *Tuteur, W., et al.* 339
Contingency Theory of Adaptation and Maladaptation. *Whittier, J. R.* 343
Evaluation of Frenquel in the Treatment of Psychotic Veterans. *Barber, R. M.* .. 345
A New Anticonvulsant, Elipten^{R}. *Lambros, V. S.* 349

SEPTEMBER

*Thyroid Therapy of Schizophrenia. *Danziger, L.* 373
Psychoanalytic Applications to Levels of Group Psychotherapy with Adults. *Kotkov, B.* 379
Reduction of EST Induced Fractures with Pentothal-succinylcholine Anesthesia. *Newbold, H. I.* 385
Some Aspects of Ego Functioning as Reflected in Skin Temperature Change. *Kanter, S. S., et al.* 388
*A New Antiphobic Agent, Benactyzine. *Feinblatt, T. M., et al.* 394
Early and Late Response to Thorazine Therapy. *Conran, P.* 399
Electroshock Therapy in the Presence of Retinal Detachment. *Karliner, W.* 401

Thyroid Therapy of Schizophrenia

Thyroid extract or thyroxine with vitamins, given to 120 schizophrenic patients, none of whom was recognized to have periodic catatonia, resulted in the recovery of 54. The recovery percentage in 80 patients given thyroid therapy for at least 100 days, which is considered to be the shortest time which can be called adequate, was 67·5. Of these 80 adequately treated patients, 60 received some form of conventional therapy (usually shock) in addition to thyroid extract or thyroxine. The immediate results are superior to those attained with adequate shock therapy in a public hospital or with various conventional methods in a private hospital.

Of 23 patients given thyroid therapy for 100 days or more after an illness of less than six months, all recovered. Of 43 patients treated for 100 days or more after an illness of less than two years, 39 (90·7 per cent.) recovered. Fifteen of the 80 patients (or 19 per cent.) recovered without requiring any shock therapy, and two of these 15 had been ill for five years or more. Those thyroid patients who received shock therapy required much less of it than had to be given to control patients who received no thyroid medications.

Relapses are known to have occurred after 24 of the 54 recoveries, but each relapse was preceded by a period of time in which the patient failed to follow directions given him as to necessary medications and laboratory tests. The incidence of relapse among those who follow directions after discharge is believed to be very small.

(Author's Abstr.)

A New Antiphobic Agent, Benactyzine

In conformity with suggestions in the American literature, and contrary to that reported by the European literature, benactyzine has been found to be an antiphobic and mild antidepressant drug but without any skeletal-muscle-relaxing effect. There are reports of slight sedative action and of slight muscle relaxation qualities (relief of night cramps).

Some physiological by-effects have been reviewed and found to be mild and relatively infrequent.

Toxicity, in the dose indicated, has not been a serious factor in clinical use.

(Authors' Abstr.)

VOL. 9 EEG CLIN. NEUROPHYSIOL. NOVEMBER, 1957

Cortical D.C. Changes Incident to Midline Thalamic Stimulation. *Goldring, S., and O'Leary, J. L.* 577
Differential Electroencephalographic Response to Conditioned Auditory Stimuli in Arousal from Sleep. *Rowland, V.* 585
Electrographic Activity of the Mesencephalic Reticular Formation During Conditioning in the Cat. *Yoshii, N., et al.* 595
Cortical and Subcortical Electrograms in Anesthesia and Anoxia in Man. *Okuma, T., et al.* 609
*Correlation of Rhinencephalic Electrograms with Behavior. *Monroe, R. R., et al.* .. 623
The Electroencephalogram and Mental Activity. *Mundy-Castle, A. C.* 643
*EEG Frequency-Pattern Variation and Intelligence. *Ellingson, R. J., et al.* 657
The Effect of Chlorpromazine and Reserpine on Sedation and Convulsive Thresholds in Schizophrenic Patients. *Bradley, P. B., and Jeavons, P. M.* 661
*Cyclic Variations in EEG During Sleep and Their Relation to Eye Movements, Body Motility, and Dreaming. *Dement, W., and Kleitman, N.* 673
Some Observations on Lambda Waves and Peripheral Stimulation. *Green, J.* .. 691

Correlation of Rhinencephalic Electrograms with Behaviour

Six patients with chronically implanted intracranial electrodes were studied under the influence of d-LSD-25, l-LSD-25, mescaline, and correlations attempted between the electrograms and behavioural observations.

An increase in beta activity and a disappearance of alpha characterized in both cortical and subcortical recordings seemed to be associated with anxiety or its derivatives.

Paroxysmal activity induced in the hippocampal, amygdaloid, and septal regions seemed to be associated with overt expressions of disturbed psychotic behaviour.

Spread of such paroxysmal activity until it became generalized in the cortex seemed to interfere with the full expression of the psychotic behaviour.

Chlorpromazine not only proved an effective blocking agent as far as the behaviour was concerned, but also abolished or minimized the appearance of the low amplitude fast activity as well as the paroxysmal subcortical activity. The possible sites of action of LSD-25, mescaline, and chlorpromazine are discussed with reference to the problems of psychomotor epilepsy, episodic psychotic reactions, and schizophrenia.

(Authors' Abstr.)

EEG Frequency-Pattern Variation and Intelligence

An experiment was performed to test a hypothesis, suggested by Walter's work, that there is a relationship between test intelligence and EEG frequency-pattern variability. This involved the application of the coefficient of concordance (Kendall's W) to frequency analyser data to yield a single summary measure of frequency-pattern variability. The hypothesis was not confirmed.

(Authors' Abstr.)

Cyclic Variations in EEG During Sleep and Their Relation to Eye Movements, Body Motility, and Dreaming

In 33 adults, discrete periods of rapid eye movement potentials were recorded without exception during each of 126 nights of undisturbed sleep. These periods were invariably concomitant with a characteristic EEG pattern, stage 1.

Composite histograms revealed that the mean EEG, eye movement incidence, and body movement incidence underwent regular cyclic variations throughout the night with the peaks of eye and body movement coinciding with the lightest phase of the EEG cycles. A further analysis indicated that body movement, after rising to a peak, dropped sharply at the onset of rapid eye movements and rebounded abruptly as the eye movements ceased.

Records from a large number of nights in single individuals indicated that some could maintain a very striking regularity in their sleep pattern from night to night.

The stage 1 EEG at the onset of sleep was never associated with rapid eye movements and was also characterized by a lower auditory threshold than the later periods of stage 1. No dreams were recalled after awakenings during the sleep onset stage 1, only hypnagogic reveries.

(Authors' Abstr.)

VOL. 10 FEBRUARY, 1958

Contribution à l'étude électroencéphalographique des syndromes endocriniens. *Thiebaut, F., et al.* 1
*Electroencephalograms of Normal, Full-term Newborns Immediately After Birth with Observations on Arousal and Visual Evoked Responses. *Ellingson, R. J.* .. 31
*Electroencephalographic Changes and Hyperammonemia. *Poser, C. M.* 51
*Electroencephalographic Findings in Acute Carbon Monoxide Poisoning. *Lennox, M. A., and Petersen, P. B.* 63
The Electroencephalogram in a Group of Normal Subjects at Sea Level and at 14,900 Feet. *Querol, M.* 69
A Study of the Factors Responsible for Changes in the Electroencephalogram in Chronic Pulmonary Insufficiency. *Wilson, W. P., and Sieker, H. O.* 89
*The Effect of Drugs on Arousal Responses Produced by Electrical Stimulation of the Reticular Formation of the Brain. *Bradley, P. B., and Key, B. J.* 97
Contribution à l'étude des rapports entre le potential réticulaire évoqué, l'état d'anesthésie et l'activité électrique cérébrale. *Longo, V. G., and Silvestrini, B.* .. 111
Autonomy of Alpha Rhythm Generators Studied by Multiple Channel Cross-correlation. *Walsh, E. C.* 121
Activités d'origine somesthésique évoquées sur le cortex non-spécifique du chat anesthésié au chloralose: rôle du centre médian du thalamus. *Albe-Fessard, D., and Rougeul, A.* 131

Electroencephalograms of Normal, Full-term Newborns Immediately After Birth with Observations on Arousal and Visual Evoked Responses

1. EEGs of 277 normal, full-term babies, ages 22 minutes to 125 hours, were recorded. Loud auditory stimuli were presented during sleep in 52 cases; single and repetitive light flashes 65 cases.

2. In general, previous descriptions of EEG patterns during wakefulness and sleep in newborns were confirmed. No relationship was observed in these data between EEG patterns and (*a*) type of maternal analgesia during labour, (*b*) type of maternal anaesthesia during delivery, or (*c*) length of 2nd stage of labour.

3. Loud auditory stimuli during sleep may elicit the following generalized responses with or without movement: (*a*) flattening of tracings, (*b*) K-type response (rare), or (*c*) K-type response followed by flattening of tracings. Spontaneous movement during sleep may also be followed by flattening of tracings.

4. Single light flashes to the eyes often elicit evoked potentials in the occipital area. These responses tend to differ from those observed in adults in the following ways:

(*a*) They are of more variable wave-form.
(*b*) They are of more variable amplitude.
(*c*) They show greater "fatiguability".
(*d*) They are of much longer latency (160–220 m/sec.).

5. Trains of light flashes at 2 f/sec. or faster tend to elicit responses at the beginning (ON responses) and end (OFF responses) of stimulation. "Driving" effects were observed in only 2 cases, at 2·5–3·5 f/sec.

6. The evoked response data are interpreted as reflecting the physiological immaturity of the newborn's nervous system associated with its anatomical immaturity. The major factor accounting for the long latencies of evoked responses is felt to be slow conduction in the afferent fiber tracts, but retinal and synaptic events may also be contributory. The peculiarities of wave-form and amplitude are felt to be functions of the immature visual cortex.

(Author's Abstr.)

Electroencephalographic Changes and Hyperammonemia

Seven patients suffering from a variety of clinical diseases and in stages of consciousness varying from normal alertness to deep coma were found to exhibit "triphasic" waves in their electroencephalographic tracings. All 7 had in common elevation of their blood ammonia levels.

The literature on blood ammonia and triphasic electroencephalographic waves is reviewed and the following conclusions are drawn: Elevation of the blood ammonia level may be accompanied by the presence of triphasic waves in the electroencephalographic tracing. However, the presence of triphasic waves more consistently indicates an elevation of the blood ammonia level. Since elevation of the blood ammonia level represents only one variable finding in a generalized metabolic disturbance, the presence of triphasic waves and their relative frequency in a single tracing do not correlate with the absolute level of the blood ammonia. The triphasic waves appear to bear no direct relation to the state of consciousness nor to the presence or absence of detectable hepatic disease or electrolyte disturbance.

It is suggested that the triphasic waves in these patients' electroencephalograms, together with their elevation blood ammonia levels, indicate an as yet unknown metabolic disturbance which may affect the reticular activating system and its diencephalocortical connections.

(Author's Abstr.)

Electroencephalographic Findings in Acute Carbon Monoxide Poisoning

1. EEGs were obtained on 33 patients from the first to the ninetieth day after acute carbon monoxide poisoning.

2. The EEGs were abnormal in 18 patients, the abnormality consisting of extreme slowing in 10, marked slowing in 3, slight in 1 and in 4 the slowing was confined to the temporal leads. The slowing was asymmetrical in 14 patients, the abnormality being equally frequent on left and right.

3. The incidence of EEG abnormality was highly correlated with the patients' age, duration of unconsciousness and state of consciousness. Eight per cent. of patients over 50 had an abnormal EEG; 33 per cent. of patients under 49. Ninety-five per cent. of patients unconscious in the hospital more than 7 hours had an abnormal EEG; 7 per cent. of patients unconscious less than 6 hours. All comatose or semi-comatose patients had an abnormal EEG; 25 per cent. of those with consciousness unimpaired. The incidence of EEG abnormality in the first week was considerably higher than when more than a week had elapsed between poisoning and EEG examination.

4. Of 20 patients with no or slight EEG abnormalities, 12 recovered fully and none died. Of 13 patients with markedly abnormal EEGs, 2 recovered fully and 5 died.

(Authors' Abstr.)

The Effect of Drugs on Arousal Responses Produced by Electrical Stimulation of the Reticular Formation of the Brain

Thresholds for behavioural and EEG arousal produced both by electrical stimulation of the reticular formation and by afferent (auditory) stimulation have been determined in *encéphale isolé* cat preparations. The effect of graded doses of pentobarbitone, chlorpromazine, atropine, hyoscine, physostigmine, amphetamine and *d*-lysergic acid diethylamide (LSD-25) on these thresholds were studied.

Pentobarbitone, in doses considerably smaller than the anaesthetic dose, blocked arousal responses completely, but chlorpromazine, even in large doses, caused only a slight rise in the thresholds for direct stimulation of the reticular formation. It blocked arousal produced by afferent stimulation.

Atropine, hyoscine and physostigmine produced a marked divergence between behavioural and EEG thresholds; atropine caused a rise in the EEG threshold and physostigmine a fall, whilst the behavioural threshold remained unchanged. The effects of hyoscine were similar to those of atropine except that it caused a slight rise in the threshold for behavioural arousal, consistent with a slight sedative effect.

Amphetamine caused the thresholds to fall until a dose was reached at which the preparation was fully alert, both behaviourally and in terms of the EEG activity. LSD-25 had no effect on the arousal threshold for stimulation of the reticular formation but produced a marked lowering of the threshold for arousal to afferent (auditory) stimulation.

The relation between these findings and the results of previous investigations is discussed, together with the possible sites of action of the drugs studied. Three different sites of action are postulated: (*a*) a direct action on the reticular activating system (pentobarbitone and amphetamine); (*b*) an action at the brain stem level but related to afferent collaterals entering the reticular formation (chlorpromazine and LSD-25); and (*c*) an action not directly related to the reticular formation of the brain stem but possibly to diffuse thalamocortical projections (atropine and physostigmine).

(Authors' Abstr.)

MAY

The Mechanism of Transition of Interictal Spiking Foci into Ictal Seizure Discharges. *Ralston, B. L.* .. 217
Single and Repetitively Evoked Potentials in Human Cerebral Cortex with D. C. Changes. *Goldring, S., et al.* .. 233
Analysis of the Electroencephalogram of Children by Histogram Method. *Fujimori, B., et al.* .. 241
Electroencephalographic Findings Associated with Congenital Heart Disease. *Shev, E. E., and Robinson, S. J.* .. 253
*Studies on the Cortical Pacemaker: Synchrony and Asynchrony of Bilateral Recorded Alpha and Beta Activity. *Garoutte, B., and Aird, R. B.* .. 259
Some Methods of Automatic Measurement of the Electroencephalogram. *Kozhevnikov, V. A.* .. 269
Electrocortical Reactions Associated with Conditioned Flexion Reflexes. *Beck, E. C., et al.* .. 279
The Occurrence of Low Voltage, Fast, Electroencephalogram Patterns During Behavioral Sleep in the Cat. *Dement, W.* .. 291
*Viscero-sensory and Somato-sensory Representation in the Rhincephalon. *Dunlop, C. W.* .. 297
An Analysis of Electrophysiological Events in Cerebral Structures During Ether Anesthesia. *Schlag, J., and Brand, H.* .. 305

Studies on the Cortical Pacemaker: Synchrony and Asynchrony of Bilateral Recorded Alpha and Beta Activity

1. By measuring and charting the times elapsed between peaks of normal alpha and beta waves recorded simultaneously from right and left homologous areas in 52 human subjects, an attempt has been made to deduce the probable source for rhythmic control of these normal frequencies.

2. Bilateral alpha and beta rhythms are probably under the control of a subcortical pacemaker or system of pacemakers. The possibility of transcallosal influences are not entirely ruled out, but seem unlikely under the circumstances discussed.

3. Shifting bilateral synchrony-asynchrony of these normal rhythms may be accounted for by one of two possible mechanisms:
(*a*) the cortical rhythm may partially escape from the deep control, then be forced to return to synchrony by the influence from below, or
(*b*) the primary source of the rhythm oscillates physiologically from side to side.

4. The progression of latencies in the occipito-frontal direction suggests transmission delays within the pacemaker itself, or in its deep subcortical connections.

5. It seems very likely that the reticular formation of the brain stem and the diffuse thalamic projection system, operating in series, provide the anatomical substratum for the physiological observations of the present study.

(Authors' Abstr.)

Viscero-sensory and Somato-sensory Representation in the Rhincephalon

1. A viscero-sensory and somato-sensory representation in the hippocampus of the marsupial phalanger is shown.

2. Stimulation of the stomach, electrically, mechanically and chemically modifies the hippocampal activity, with electrical stimulation eliciting the most striking results.

3. A slow wave is evoked in the hippocampus 500 m/sec. after stimulation. Where a primary response with short latency occurred, no slow wave ensued, and evidence is presented that the primary response may have resulted from stimulation of adjacent somatic structures.

4. A somato-sensory representation in the amygdala is demonstrated, with no viscero-sensory representation evident.

(Author's Abstr.)

VOL. 10 AUGUST, 1958

Sequential Alterations in the Electroencephalograms of Patients with Brain Tumors. *Daly, D. D., and Thomas, J. E.* .. 395
The Electroencephalogram in Brain Stem Vascular Disease. *Tucker, J. S.* .. 405
Electronic Analysis of Cerebral Responses to Photic Stimulation in Patients with Brain Damage. *Kooi, K. A., and Thomas, M. H.* .. 417
Re-Evaluation of Sleep Electroencephalography. *Silverman, D., and Morisaki, A.* .. 425

Metrazol Tolerance in a "Normal" Volunteer Population. *Rodin, E.* 433
Patterns of Cortical Discharges and Their Relation to Routine Scalp Electroencephalography. *Abraham, K., and Ajmone-Marsan, C.* 447
Direct Recording of Spontaneous and Evoked Seizures in Epileptics. *Delgado, J. M. R., and Hamlin, H.* 463
Conditioning of Frequency-Characteristic Repetitive Electroencephalographic Response with Intermittent Photic Stimulation. *Yoshii, N., and Hockaday, W. J.* 487
The Encephalogram and Electromyogram of Motor Conditioned Reflexes After Paralysis with Curare. *Majkowski, J.* 503
The Influence of Chlorpromazine Upon the Cerebello-Spinal Circuits. *Kreindler, A., et al.* 515
Rate of Impulse Conduction in Denervated Human Muscle. *Buchthal, F., and Rosenfalck, P.* 521
The Form of Motor-Unit and Fibrillation Potentials. *Jarcho, L. W., et al.* 527

VOL. 47 **ENCEPHALE.** 1958

EEG and Medico-legal Opinion. *Delay, J., et al.* 1
Character and Stages of the Mental Deterioration in Subacute Sclerosing Leucoencephalitis. *Hasaerts, E., and van Geertruyden* 31
EEG Study of Acute Confusional States. *Kammerer, T., et al.* 73
Amputation Neuroma and Phantom Limb. *Vereecken, J. L. T. M.* 88
An Anatomo-clinical Study of Eight Alcoholic Encephalopathies. *Delay, J., et al.* 99
Experience with LSD-25. *Delay, J., and Benda, P.* 169
Choreo-athetotic Movements and Vaso-motor Symptoms in an Imbecile. *Beley, A., and Bieder, J.* 210
The Physiopathological Bases of the Disintegration of Motor Functions in Parkinsonian Rigidity. *Barraquer-Bordas, L.* 217
Present Problems of Progressive Myoclonic Epilepsy of Unverricht-Lundborg. *Novelletto, A.* 223
Temporal Lobectomy in the Treatment of Psychomotor Epilepsy. *Alajouanine, T., et al.* 265
Experience with LSD-25 (concluded). *Delay, J., and Benda, P.* 309

VOL. 60 **FOL. PSYCHIAT. NEUR. NEUROCHIR. NEERL.** AUGUST, 1957

On Leucodystrophy and Leucoencephalitis. *de Vries, E.* 255
Psychopathology and Symptomatology of the Manic-depressive Border State. *Hutter, A.* 283
Afferent Fibres to the Intermediate and Solitary Tract Nuclei. *Sie, P. G.* 302

OCTOBER

A Contribution to the Study of the Rorschach Test in the Epilepsies. *Schachter, M.* 353
The Course of Development of a Case of Induced Psychosis. *Tolsina, F. J.* 376
Schizophrenia and Cybernetics. *Hirschfeld, H.* 388

DECEMBER

Biological, Psychological and Neuropsychiatric Observations on Oligophrenia. *Prick, J. J. G.* 435
Clinical Problems in Oligophrenic Conditions. *Kamp, L. N. J.* 463
Principles in the Psychological Examination of Oligophrenics. *Greivel, I.* 475
The Significance of Convulsions in Infants as a Cause of Cerebral Damage. *de Vries, E.* 493

VOL. 61 FEBRUARY, 1958

Lypothmia. *van Wulfften Palthe, P. M.* 1
Rorschach's Experience-Type and Jung's Attitudinal-Type. *van den Broek, P.* 55

JUNE

Problems with Regard to the Aetiology and Genesis of Mental Diseases. *Prick, J. J. G.* 297
Trigeminal Neuralgia and Arterio-venous Aneurysm of the Posterior Fossa. *Weersma, M.* 315
The Problem of Incest. *Bax, J.* 328
The C.S.F. Aspects of Leuco-encephalitis. *Booy, J.* 352
Premature Senile Dementia of Mongols. *Jelgersma, H. C.* 367

VOL. 57 **GENET. PSYCHOL. MONOGR.** FEBRUARY, 1958

Psychological and Cultural Problems in Mental Subnormality: A Review of Research. *Sarason, S. B., and Gladwin, T.* 3

MAY

Developmental Aspects of Discrimination in Relation to Adjustment. *London, P.* 293
Muscular Tension as a Response to Psychological Stress in Rheumatoid Arthritis and Peptic Ulcer. *Southworth, J. A.* 337
Frustration: Theory and Experiment. *Lawson, R., and Marx, M. H.* 393

VOL. 58 AUGUST, 1958

Emotional Aspects of Political Behavior: The Woman Voter. *Bennett, E. M., and Goodwin, H. M.* 3

The Accuracy of Self Estimate: A Measure of Self-concept Reality. *Brandt, R. M.* 55

Personality Factors in Social Mobility: A Study of Occupationally Mobile Business Men. *Abegglen, J. C.* 101

VOL. 85 **GIORN. PSICHIAT. NEUROPAT.** 1957

Psychodynamic Mechanisms in Hallucinatory Delusional Syndromes of Schizophrenic Type. *Floris, V., and Della Rocca, G.* 751

Psychodynamic Considerations About a Case of Genealogical Delirium. *Vizioli, R., et al.* 791

On Mental Dissociation. *Bambini, G., and Sanguinetti, A.* 813

Hydrocephalus in Children. *Cavazzuti, G. B., and Canossi, G. C.* 827

Nocturnal Epilepsy in Children. *Morocutti, C., and Vizioli, R.* 875

Clinical Contribution to the Localizing and Pathogenic Question of Retraction Nystagmus. *Saginario, M.* 895

Muscular Atrophies and Fibrillations of Central Origin. *Migliore, A., and Kapleone, F.* 925

The Influence of the Cerebrospinal Fluid on Plasma Coagulation. *Bovi, A.* 935

The Importance of Hands in Mind Formation. *Neri, V.* 975

Considerations on the Treatment of Thomsen's Disease and Steinert's Myotonic Dystrophy with Prednisone. *Scarzella, R.* 1001

Considerations and Statistical Remarks about 427 Cases of Transorbital Leucotomy. *Cenaechi, G.* 1015

VOL. 86 1958

The Drawing of a Tree as a Means of Phenomenological Research on the Structure of Space in Mental Patients. *Gomirato, G., et al.* 1

Investigations into the Humoral Syndrome of Mongolism. *Torre, M.* 17

An Angiographic Study of 207 Surgically Verified Supratentorial Meningiomas. *Bernasconi, V., and Cassinari, V.* 63

Further Observations on Frenquel. *Puca, A., and Catapano, V. D.* 115

O-antistreptolysin Titre in Serum of Psychoneurotics. *Andreani, G.* 139

Determination of the Amount of Serum Copper in Schizophrenic Subjects. *Bovi, A., and Mascellani, A.* 163

Usual and Unusual EEG Experiences Concerning Cerebral Pathology. *Battisti, A., et al.* 175

EEG Observation on a Case of Narcolepsy. *Mantelli, G.* 221

Schizophrenic and Galvanic Skin Response. *Missaglia, A.* 231

The Use of Lithium Salts in the Treatment of Mental Diseases. *Andreani, G., et al.* 273

Clinical and EEG Experiences on the Association of Chlorpromazine and Reserpine. *Caselli, G., and Mantelli, G.* 329

The Drawing of a Tree Applied to the Study of the Psychopathological Modifications Caused by LSD-25 in Schizophrenia. *Gomirato, G., et al.* 433

Features of the Drawing of the Tree in Mentally Abnormal People. *Gomirato, G., and Gamra, G.* 485

Present Status of the Medical Therapy of Epilepsy. *Rigotti, S., and Schergna, E.* 497

Psychological Study with the Rorschach Test on a Triplet Twin Sisters. *Schachter, M.* 541

Results of the H Responses to the Rorschach Test in 12–16-year-old Children. *Cotte, S.* 553

Fear as a Critical Phenomenon in Injuries of the Gyrus Cinguli. *Alvisi, G., and Ballerini, A.* 561

O-antistreptolysin Titre in Tics of Childhood, Mental Diseases, and Nervous Diseases. *Andreani, G.* 575

Confusional State in Monozygotic Twins. *Rigotti, S., and Terzian, H.* 673

Anatomical and Clinical Contribution to the Study of Neurinomas of the Spine. *Schiffer, D.* 687

Observations on a Cerebral Granuloma Produced by Blastomyces. *Alrisi, C., and Giordano, G. B.* 703

Progressive Paralysis of Old Age. *Mancini, E., et al.* 721

The Language of Hands in Insanity. *Neri, V.* 741

A Contribution to the Interpretation of Transitory Motor Troubles Following Shock Therapies. *Accornero, F., and Guerra, A.* 761

The "Tree Test of Koch" in the Tics of Childhood. *Andreani, G., and Bovi, A.* 781

Explosive Laugh Fits as a First Manifestation of Temporal Epilepsy. *Martelli, G.* 799

The Test of (Tsedek) Baruk in the Tics of Childhood. *Andreani, G., and Bovi, A.* 813

VOL. 39 **INTERNAT. J. PSYCHO-ANAL.** MARCH-AUGUST, 1958

Toward an Understanding of the Physical Nucleus of Some Defence Reactions. *Greenacre, P.* 69

Autism and Symbiosis, Two Extreme Disturbances of Identity. *Mahler, M. S.* 77

On the Development of Mental Functioning. *Klein, M.* 84

Bisexuality and Ego Structure. *Weiss, E.* 91

The Allergic Object Relationship. *Marty, P.* 98
Peptic Ulcer and Pseudo Peptic Ulcer. *Garma, A.* 104
Noise, Speech and Technique. *Scott, W. C. M.* 108
On the Preoedipal Phase of the Male. *Van der Leeuw, P. J.* 112
The Two Genetic Derivations of Aggression with Reference to Sublimation and Neutralization. *Lantos, B.* 116
Some Observations on the Psychopathology of Hypochondriacal States. *Rosenfeld, H.* 121
The Manifold Possibilities of Therapeutic Evaluations of Dreams. *Kemper, W.* 125
On the Process of Psycho-analytical Inference. *Money-Kyrle, R. E.* 129
On Delusional Transference (Transference Psychosis). *Little, M.* 134
Sterility and Envy. *Langer, M.* 139
On Arrogance. *Bion, W. R.* 144
Magic and the Castration-Complex. *Servadio, E.* 147
A Screen Memory and Myth Formation in a Case of Apparent Precognition. *Székely, L.* 151
Some Thoughts on Postpartum Respiratory Experiences and Their Relationship to Pregenital Mastery, Particularly in Asthmatics. *Bell, A. I.* 159
Dentition, Walking, and Speech in Relation to the Depressive Position. *Pichon-Riviere, A.* 167
Ego Reintegration Observed in Analysis of Late Adolescents. *Adatto, C. P.* 172
Fear of Death. Notes on the Analysis of an Old Man. *Segal, H.* 178
Struggle Against the Introjects. *Bychowski, G.* 182
Maternal Narcissism and the Oedipus Complex. *Hart, H. H.* 188
The Ego and the Function of Ideology. *Baranger, W.* 191
Psychodynamics of Motility. *Mittelmann, B.* 196
Variations in Classical Psycho-analytic Technique: An Introduction. *Greenson, R. R.* 200
Remarks on Some Variations in Psycho-analytic Technique. *Loewenstein, R. M.* 202
Technical Variation and the Concept of Distance. *Bouvet, M.* 211
Remarks on Some Variations in Psycho-analytical Technique. *Eissler, K. R.* 222
A Special Variation of Technique. *Reich, A.* 230
Variations in Technique. *Nacht, S.* 235
Contribution to the Discussion on Variations in Classical Technique. *Rosenfeld, H.* 238
Variations in Classical Technique: Concluding Remarks. *Loewenstein, R. M.* 240
Neurotic Ego Distortion: Opening Remarks to the Panel Discussion. *Waelder, R.* 243
On Ego Distortion. *Gitelson, M.* 245
Neurotic Ego Distortion. *Gillespie, W. H.* 258
Ego-Distortion. *Glover, E.* 260
Contribution to the Panel on Ego Distortion ("As-if" and "Pseudo As-if"). *Katan, M.* 265
Causes and Mechanisms of Ego Distortion. *Nacht, S.* 271
Discussion on Ego Distortion. *Rosenfeld, H.* 274

VOL. 56 J. AB. SOC. PSYCHOL. JANUARY, 1958

Clinical and Statistical Prediction: A Reformulation and Some New Data. *Holt, R. R.* 1
Social Influence Phenomena in a Task-Oriented Group Situation. *Kidd, J. S.* 13
Personality and Psychopathology. *Burdock, E. I., et al.* 18
The Psychodynamics of Social Mobility in Adolescent Boys. *Douvan, E., and Adelson, J.* 31
The Relation of Achievement Motivation to Problem-Solving Effectiveness. *French, E. G., and Thomas, F. H.* 45
The Effect of Brief Social Deprivation on Behaviors for a Social Reinforcer. *Gewirtz, J. L., and Baer, D. M.* 49
Preference for Delayed Reinforcement: An Experimental Study of a Cultural Observation. *Mischel, W.* 57
Empathy or Behavioral Transparency? *Foa, U. G.* 62
Rigidity, Insecurity and Stress. *Ainsworth, L. H.* 67
Intelligence and Emotional Disturbance: Evidence from Army and Veterans Administration Records. *Miner, J. B., and Anderson, J. K.* 75
Influencing Ethnocentrism in Small Discussion Groups Through a Film Communication. *Mitnick, L. L., and McGinnies, E.* 82
Some Factors in Self-Disclosure. *Jourard, S. M., and Lasakow, P.* 91
Two Forms of Social Conformity: Acquiescence and Conventionality. *Beloff, H.* 99
The Role of Motivation in Psychological Stress. *Vogel, W., et al.* 105
Interpersonal Preference and Cognitive Organization. *Kogan, N., and Tagiuri, R.* 113

MARCH

Is the F Scale Irreversible? *Christie, R., et al.* 143
Differential Autonomic Responsiveness in the Process-Reactive Classification of Schizophrenia. *King, G. F.* 160
Ambiguity and Anxiety. *Dibner, A. S.* 165
The Application of Learning Theory to the Treatment of Tics. *Yates, A. J.* 175
GSR Conditioning in Anxiety States, Normals, and Chronic Functional Schizophrenic Subjects. *Howe, E. S.* 183
Parental Evaluations of Children and Children's Self-Evaluations. *Helper, M. M.* 190
Identification in Group Therapy. *Briskin, G. J.* 195

Some Relationships Among Projective Test, Interview, and Sociometric Measures of Dependent Behavior. *Fitzgerald, B. J.* .. 199
The Repression Hypothesis Studied in a Situation of Hypnotically Induced Conflict. *Bobbitt, R. A.* .. 204
The Effect of Sibling Competition and Alliance on Level of Aspiration, Expectation, and Performance. *Ferguson, E. D.* .. 213
Effects of Public and Private Failure on Self-Evaluation. *Stotland, E., and Zander, A.* 223
Distance as a Determinant of Influence in a Two-Person Serial Interaction Situation. *Fisher, S., and Lubin, A.* .. 230
The Influence of Language-Acquisition Contexts on Bilingualism. *Lambert, W. E., et al.* 239
Obligations to Self and Society in the United States and Germany. *McClelland, D. C., et al.* .. 245
Sex Differences in Problem-Solving Performance as a Function of Attitude Differences. *Carey, G. L.* .. 256
Personality and I.Q. Change. *Kagan, J., et al.* .. 261
The Psychology of Smoking. *McArthur, C., et al.* .. 267

MAY

The Measurement of Preconscious Perception in Dreams and Images: An Investigation of the Poetzl Phenomenon. *Shevrin, H., and Luborsky, L.* .. 285
Affective Response to Psychoanalytic Death Symbols. *Meissner, W. W.* .. 295
The Specificity of Intolerance of Ambiguity Measures. *Kenny, D. T., and Ginsberg, R.* 300
Perceptual Reactivity to Threat and Neutral Words Under Varying Experimental Conditions. *Cowen, E. L., and Obrist, P. A.* .. 305
Authoritarianism, Punitiveness, and Perceived Social Status. *Roberts, A. H., and Jessor, R.* .. 311
Conformity and Problem Solving. *Nakamura, C. Y.* .. 315
A Restatement and Test of Schlosberg's Theory of Emotion With Two Kinds of Subjects from Greece. *Triandis, H. C., and Lambert, W. W.* .. 321
Psychological Test and Organismic Correlates of Interview Interaction Patterns. *Matarazzo, R. G., et al.* .. 329
Interrelationships Among Individual Difference Variables, Behavior in Psychotherapy, and Verbal Conditioning. *Sarason, I. G.* .. 339
Interrelations Among Measures of Personality Adjustment in Nonclinical Populations. *Fiedler, F. E., et al.* .. 345
Interacting Effects of Control, Motivation, Group Practice, and Problem Difficulty on Attempted Leadership. *Bass, B. M., et al.* .. 352
Effects of Motivating Instructions on Reaction Time in Cerebral Disease. *Blackburn, H. L.* .. 359
Autonomic Feedback: The Perception of Autonomic Activity. *Mandler, G., et al.* .. 367
Intrusion of Associative Responses Into Schizophrenic Conceptual Performance. *Chapman, L. J.* .. 374
The Relationship Between Judgments of a Communicator's Position and Extent of Opinion Change. *Weiss, W.* .. 380

VOL. 57 JULY, 1958

A Microgenetic Approach to Word Association. *Flavell, J. H., et al.* .. 1
Improvement in the Performance of Schizophrenics on Concept Formation Tasks as a Function of Motivational Change. *Cavanaugh, D. K.* .. 8
Some Clinical Correlates of Operant Behavior. *Mednick, M. T., and Lindsley, O. R.* 13
The Effect of Person-Group Relationships on Conformity Processes. *Jackson, J. M., and Saltzstein, H. D.* .. 17
Variability of Light Perception Thresholds in Brain-Injured Children. *Mark, H. J., and Pasamanick, B.* .. 25
The Effect of Subliminal Stimulation Upon Autonomic and Verbal Behavior. *Dixon, N. F.* .. 29
Attitudes Established by Classical Conditioning. *Staats, A. W., and C. K.* .. 37
Changes in Religious Interest: A Retest After 15 Years. *Bender, I. E.* .. 41
Attempts to Lead, Group Productivity, and Morale Under Conditions of Acceptance and Rejection. *Pepinsky, P. N., et al.* .. 47
The Effects of Anxiety Level and Psychological Stress on Verbal Learning. *Taylor, J. A.* 55
Patterns of Managerial Traits and Group Effectiveness. *Ghiselli, E. E., and Lodahl, T. M.* 61
Thematic Apperception Test: An Empirical Examination of Some Indices of Homosexuality. *Lindzey, G., et al.* .. 67
Thematic Apperception Test: Some Evidence Bearing on the "Hero Assumption". *Lindzey, G., and Kalnins, D.* .. 76
Reinforcement of Affect Responses of Schizophrenics During the Clinical Interview. *Salzinger, K., and Pisoni, S.* .. 84
The Effects of Punishment (Electric Shock) on Perceptual Learning. *McNamara, H. J., et al.* .. 91

Intralist Similarity and Verbal Rote Learning Performance of Schizophrenic and Cortically Damaged Patients. *Carson, R. C.* 99

The Intelligence Test Performance of Maori Children: A Cross-Cultural Study. *Walters, R. H.* 107

SEPTEMBER

The Acceptance of Information Into Attitude Structure. *Peak, H., and Morrison, H. W.* 127

Personality Variables and Recognition Response Level. *Binder, A.* 136

Hysterics and Dysthymics as Criterion Groups in the Study of Introversion-Extraversion. *Sigal, J. J., et al.* 143

Facial Expression and Situational Cues. *Frijda, N. H.* 149

The Organizing Function of Interaction Roles in Person Perception. *Jones, E. E., and deCharms, R.* 155

Deprivation and Satiation of Social Reinforcers as Drive Conditions. *Gewirtz, J. L., and Baer, D. M.* 165

The Effects of Leadership Style and Leadership Power Upon the Inducement of an Attitude Change. *Kipnis, D.* 173

Social Role and Patterns of Symptomatic Behaviors. *Phillips, L., and Rabinovitch, M. S.* 181

A Comparative Factorial Analysis of Semantic Structures in Monolingual Greek and American College Students. *Triandis, H. C., and Osgood, C. E.* 187

Conditioning of Attention as a Source of Autistic Effects in Perception. *Walters, R. H.* 197

Self-Ideal Congruence as an Index of Adjustment. *Turner, R. H., and Vanderlippe, R. H.* 202

Some Effects of Feedback from the Experimenter on Conformity Behavior. *Jones, E. E., et al.* 207

The Role of Social Experience in the Production of Gastric Ulcers in Hooded Rats Placed in a Conflict Situation. *Conger, J. J., et al.* 214

VOL. 14 **J. CLIN. PSYCHOL.** JANUARY, 1958

The Character and Degree of Psychopathology in Relation to Sustained Intellectual Performance. *Roessler, R., and Roberts, L.* 3

Spontaneous Fluctuations in the Severity of Illness of Hospitalized Lobotomized Patients. *Hanlon, T. E., et al.* 6

Differences in Some Personal Characteristics of "Rigid" and "Flexible" Individuals. *Schaie, K. W.* 11

Further Studies on the Bender-Gestalt Test and the Digit Span Test as Measures of Recall. *Tolor, A.* 14

Age, Intelligence, Aphasia and the Spiral After-Effect in an Epileptic Population. *Aaronson, B. S.* 18

Wechsler-Bellevue Changes over Time. *Armitage, S. G., and Pearl, D.* 22

A Comparison of the Bender-Gestalt Reproductions of Delinquents and Non-Delinquents. *Zolik, E.* 24

PEAQ: A Measure of Psychopathic Behavior. *Applezweig, M. H., et al.* 26

The Hill-Monroe Inventory for Predicting Acceptability for Psychotherapy in the Institutionalized Drug Addict. *Monroe, J. J., and Hill, H. E.* 31

A New Sorting Technique for Diagnosing Brain Damage. *Altrocchi, J., and Rosenberg, B. G.* 36

An Evaluation Scale for Shock Therapy. *Feldman, M. J.* 41

A Validation of Measures that Predict the Efficacy of Shock Therapy. *Pumroy, D. K., and Kogan, W. S.* 46

An Evaluation of the Grassi Test for Organic Involvement. *Hirt, M.* 48

An Evaluation of the "Organic Signs" in the H-T-P Drawings. *Bieliauskas, V. J., and Kirkham, S. L.* 50

Human Figure Drawings by Younger and Older Adults. *Lorge, I., et al.* 54

Manifest Content and Interpretive Meaning of Verbal Intelligence Test Responses. *Tallent, N.* 57

Rorschach *M* Responses and Intelligence. *Sommer, R.* 58

Global Evaluation of Rorschach Performance Versus Scores: Sex Differences in Rorschach Performance. *Richards, T. W., and Murray, D. C.* 61

Utilization of O'Reilly's Objective Rorschach as a Screening Test for State Colony Job Applicants. *Murphy, M. M.* 65

Movement in the Fantasy of Brain-Injured (Cerebral Palsy) Children. *Richards, T. W.* 67

Differentiating Alcoholics from Normals and Abnormals with the MMPI. *Hoyt, D. P., and Sedlacek, G. M.* 69

The Effect on Self Description of Combining Anxiety and Hostility Items on a Single Scale. *Feldman, M. J., and Siegel, S. M.* 74

Face Validity in the *D* Scale of the MMPI. *McCall, R. J.* 77

The Taylor Manifest Anxiety Scale in Differential Diagnosis. *Rubin, H., and Townsend, A. H.* 81

Prediction of Rehospitalization Using the MMPI. *Briggs, P. F.* 83

Note on a Rapid Scoring Procedure for the Card Form MMPI. *Wilcox, G. T.* 85

The Clinical Application of Sequential Analysis. *Trehub, A., and Scherer, I. W.* 86

An Approximation Table for Determining Sample Size by Means of the Sign Test. *Rawn, M. L.* 90
Construction of a Table of the Significance of the Difference between Verbal and Performance I.Q.s on the WAIS and the Wechsler-Bellevue. *Wolfensberger, W. P.* 92
The Effect of a Recorded Voice on the Drawing of a Geometric Design as a Predictor of Social Behavior. *Shear, H. J.* 93
A Note on the Reliability of the L-M Fergus Falls Behavior Rating Scale. *Sines, L. K.* 95

APRIL

Personality and Creativity in Artists and Writers. *Drevdahl, J. E., and Cattell, R. B.* 107
The Psychologically Healthy Person: A Study in Identification and Prediction. *Schultze, K. V.* 112
A Comparison of Some Concepts of Psychological Health. *Mehlman, B., and Kaplan, J. E.* 118
Life Record Criteria of Psychological Health. *Thorne, F. C.* 123
A Factor Analytic Study of Anxiety. *Martin, B.* 133
A Brief Scale for Juvenile Delinquency. *Quay, H., and Peterson, D. R.* 139
A Comparison of Action Patterns of Schizophrenic and Normal Adults. *Friedman, H.* 142
The Differentiation of Schizophrenic and Superficially Similar Reactions. *Harding, G. F., et al.* 147
The Constriction-Dilatation Dimension in Rorschach and TAT. *Shatin, L.* 150
Response Time and Rorschach Behavior. *Hafner, A. J.* 154
A Comparison of Rorschach and Howard Ink Blot Tests on a Schizophrenic Population from a Content Point of View. *Scott, E. M.* 156
Examiner, Ego Defense, and the H-T-P. *Cassel, R. H., et al.* 157
A Comparison of H-T-P Responses of Handicapped and Non-Handicapped Children. *Wawrzaszek, F., and Johnson, O. G.* 160
Superiority-Inferiority Index in Relation to Frustrating Situations. *Foulds, G. A.* .. 163
Security-Insecurity and the Direction of Aggressive Responses to Frustration. *Bennett, C. M., and Jordan, T. E.* 166
Sexual Symbolism in Line Qualities. *Glatter, A. N., and Hauck, P.* 168
Sexual Differentiation or Artistic Ability? *Sherman, L. J.* 170
A Re-evaluation of the Color Drawing Test. *Tolor, A.* 172
Empathy and Its Relation to Stimulus Meaning. *Lair, C. V.* 175
Judgments of Personality Traits in the Self and Others. *Krevelen, A. V.* 178
An Experimental Investigation of the Psychodynamic Implications of the Hypnotic "Previous Experience" Fantasy. *Zolik, E. S.* 179
Attitudes of Parents of Hospitalized Children Towards Doctors, Nurses and Husbands: The Construction of Three Scales. *Murstein, B. I.* 184
Personality Factors and Epileptic Seizures. *Graham, L. R.* 187
The Abbreviated Wechsler Adult Intelligence Scale in a Psychiatric Population. *Whitmyre, J. W., and Pishkin, V.* 189
Wechsler-Bellevue Intelligence as a Predictor of Continuation in Psychotherapy. *Hiler, E. W.* 192
Prediction of College Performance by Commonly Used Tests. *Sopchak, A. L.* .. 194
Performance of Two Types of Depressives on a Test of Symbol Arrangement. *Kahn, T. C.* 197
The Role of the Nurse in Psychological Testing. *Sommer, R.* 200
Exceptional Memory for Dates and Weather in a Schizoid Psychopath. *Robertson, J. P. S.* 202
A Comparison Between Normals and Mental Patients of the Perception of Sexual Symbols. *Glatter, A. N., and Hauck, P.* 204
A Note on Scoring Recalled Figures of the Bender Gestalt Test Using Psychotics, Non-psychotics and Controls. *Stewart, H., and Cunningham, S.* 207
Buffer Item Variation and Schizophrenic Anxiety Scores. *Lebo, D., and Noblin, C. D.* 209

JULY

The Performance of Psychiatrists and Psychologists in a Therapeutic Interview. *Strupp, H. H.* 219
Criteria of Therapeutic Response in Hospitalized Psychiatric Patients. *Schofield, W., and Briggs, P. F.* 227
The Reliability of Clinical Judgments of Asocial Tendency. *Hunt, W. A., and Jones, N. F.* 233
Clinical Judgment of Some Aspects of Schizophrenic Thinking. *Hunt, W. A., and Jones, N. F.* 235
Judging Rorschach Responses: An Illustrative Protocol. *Hamlin, R. M., and Powers, W. T.* 240
On Individualizing the Psychologist's Clinical Evaluation. *Tallent, N.* 243
A Mental Hygiene Clinic Case Survey. *Feldman, R., et al.* 245
Comparison of Three Methods of Handling Mental Hygiene Clinic Statistics. *Rogers, L. S.* 251
Psychological Reports Related to Behavior and Benefit of Placement in Home Care. *Ullmann, L. P., et al.* 254

Psychiatric Patient Audience Reactions to Types of Motion Pictures. *Whitmyre, J. W.* 259
Ambiguities in the Role of Clinical Psychologist in a Mental Hospital. *Sommer, R., and Clancy, I. L. W.* 264
A Validation Study of the Custodial Mental Illness Ideology Scale. *Rogers, A. H., et al.* 269
Admission Rates to a State Hospital: 1920–1956. *Hauck, P. A., and Hogue, T.* .. 271
The Performance of High-Grade Retarded, Emotionally Disturbed Children on the Children's Manifest Anxiety Scale and Children's Anxiety Pictures. *Rosenblum, S., and Callahan, R. J.* 272
The Role of Anxiety in Psychodiagnosis: Replication and Extension. *Ullmann, L. P., and Hunrichs, W. A.* 276
The Overt-Covert Anxiety Index and Hostility. *Rawn, M. L.* 279
The Influence of Instructional Set upon the Discomfort-Relief Quotient. *Lebo, D., and Applegate, W.* 280
Relationships Between the Adjective Check-List, Personal Preference Schedule and Desirability Factors under Varying Defensiveness Conditions. *Heilbrun, A. B., Jr.* 283
Survey of Painfulness Concepts. *Petrovich, D. V.* 288
The Bender Gestalt Test and Learning Disturbances in Young Children. *Koppitz, E. M.* 292
Judging Intellectual Status from the Bender-Gestalt Test. *Peek, R. M., and Storms, L. H.* 296
Effects of Hypophysectomy on Bender-Gestalt Test Performance. *Schon, M., and Waxenberg, S. E.* 299
Reading Failure as a Factor in the WAIS Subtest Patterns of Youthful Offenders. *Graham, E. E., and Kamano, D.* 302
MMPI Profile Configurations among Crime Classification Groups. *Panton, J. H.* .. 305
Predicting Prison Adjustment with the Minnesota Multiphasic Personality Inventory. *Panton, J. H.* 308
Second-Order Personality Factors and the MMPI. *Karson, S.* 313
A Validation of MMPI Scale Pairs in Psychiatric Diagnosis. *Taulbee, E. S.* 316
Reliability-Stability of Subtest Scatter on the Wechsler-Bellevue Intelligence Scales. *Griffith, R. M., and Yamahiro, R. S.* 317
Dental Caries and Psychological Factors. *Manhold, J. H., Jr., and Hafner, A. J.* .. 319
Role-Taking Ability and Schizophrenia. *Diamond, M. D.* 321
Stereotypes of Nationalities by Student Nurses. *Graham, T. F.* 324
The Maturational Factor in Humor. *Graham, L. R.* 326
Recovery from Paranoid Delusions Following Hypnotic Uncovering of Repressed Episodes. *Stauffacher, J. C.* 328

OCTOBER

Reproducible Personality Factors in Middle Childhood. *Coan, R. W., and Cattell, R. B.* 339
Personality Factors in Nursery School Children as Derived from Parent Ratings. *Peterson, D. R., and Cattell, R. B.* 346
Personality Factors in the Readiness to Express Aggression. *Worchel, P.* 355
The Relationship between the Self Concept and the Ideal-Self Concept as a Measure of Adjustment. *Nahinsky, I. D.* 360
The Self Concept in Paranoid Schizophrenia. *Rogers, A. H.* 365
The Pain Apperception Test: Psychological Correlates of Pain Perception. *Petrovich, D. V.* 367
Graphic Representation of Emotional Feelings. *Peters, G. A., and Merrifield, P. R.* .. 375
The Validity of the Edwards Personal Preference Schedule in the Measurement of Dependency-Rebelliousness. *Zuckerman, M.* 379
Manifest Anxiety and Mother-Son Identification. *Stewart, L. H.* 382
The Psychasthenic and Hypomanic Scales of the MMPI and Uncertainty in Judgments. *Griffith, A. V., et al.* 385
Comparability of MMPI Card and Booklet Forms for Psychiatric Patients. *Rosen, A., et al.* 387
A Note on the Effect on Self-Description of Combining Anxiety and Hostility Items on a Single Scale. *Siegel, S. M., and Feldman, M. J.* 389
Scores of Alcoholics on the Sixteen Personality Factor Questionnaire. *De Palma, N., and Clayton, H. D.* 390
The Leary Interpersonal Check List: A Reliability Study. *Armstrong, R. G.* 393
Selective and Differentially Accelerated Intellectual Dysfunction in Specific Brain Damage. *Fisher, G. C.* 395
Intelligence Test Correlates of Shipley-Hartford Performance. *Sines, L. K.* 399
A Proposed Method for Measuring Potential Intelligence. *Mahrer, A. R., and Bernstein, L.* 404
Inference of Intellectual Efficiency from the WAIS Vocabulary Subtest. *Fink, S. L., and Shontz, F. C.* 409
Relationships between the Bender Gestalt Test and the Wechsler Intelligence Test for Children. *Koppitz, E. M.* 413
A Multiple Choice Supraordinality Test. *Peters, H. N.* 416
An Experimental Study of Transference and Resistance Phenomena in Psychoanalytically Oriented Psychotherapy. *Rawn, M. L.* 418

Insight and Prognosis. *Eskey, A.* 426
A Comparative Judgmental Study of "Defection" from Treatment at a Child Guidance Clinic. *Levitt, E. E.* 429
Alternating Two-Day Cyclic Behavior Changes. *Weiss, A. A.* 433

VOL. 108 J. COMP. NEUR. DECEMBER, 1957

Spino-vestibular Fibers in the Cat. An Experimental Study. *Pompeiano, O., and Brodal, A.* 353
The Effects of Auditory Pathway Interruption on the Incidence of Sound-Induced Seizures in Rats. *Koenig, E.* 383
The Behavior of the Epibranchial Placode of the Facial Nerve in the Sheep. *Batten, E. H.* 393
Studies on the Innervation of the Bone Marrow. *Takase, B., and Nomura, S.* .. 421
Structural Variations of Nerve Endings in the Striated Muscles of the Rat. *Cole, W. V.* 445
Descending Connections to the Vestibular Nuclei. An Experimental Study in the Cat. *Pompeiano, O., and Walberg, F.* 465
*Rhinencephalic Lesions and Behavior in Cats. An Analysis of the Klüver-Bucy Syndrome with Particular Reference to Normal and Abnormal Sexual Behavior. *Green, J. D., et al.* 505

Rhinencephalic Lesions and Behaviour in Cats. An Analysis of the Klüver-Bucy Syndrome with Particular Reference to Normal and Abnormal Sexual Behaviour

1. In a series of 122 cats, lesions were placed in the amygdala-pyriform cortex and in other rhinencephalic areas in 82. Forty animals were used as controls.
2. Observed variations in normal sex behaviour and sex behaviour following hormone administration are described.
3. Sex hormones have behavioural effects on prepubertal animals specific to the type of hormone.
4. Sex hormones have behavioural effects in adults depending on the sex of the animal or previous conditioning, although their effects on target organs depend on the hormone.
5. Male hypersexual behaviour may be induced by lesions restricted to the pyriform cortex.
6. This effect (5) is not due simply to a raised testosterone titre.
7. Hyperphagia has been produced by lesions restricted to the basal and lateral part of the amygdala.
8. Seizures observed in some animals were closely correlated with fear-like behaviour and damage to the hippocampus.
9. Cataleptic changes were associated with lesions involving the rostral amygdala and basal ganglia.
10. Other phenomena observed were: hyperthermia, emotional changes and oral behaviour. These are discussed.
11. It is suggested that many of the phenomena associated with stimulation or destruction of the pyriform cortex may be secondary to the hallucinations and fugues of psychomotor epilepsy, though other changes are difficult to explain on this basis.

(Authors' Abstr.)

VOL. 109 FEBRUARY, 1958

Morphological Observations on the Cerebellar Cortex. *Braitenberg, V., and Atwood, R. P.* 1
Myeloschisis and Myelomeningocele Produced Experimentally in the Rat. *Warkany, J., et al.* 35
*Isolated Lesions of the Fastigial Nuclei in the Cat. *Carpenter, M. B., et al.* 65
Optic Centers and Pathways After Eye Removal in Fetal Guinea Pigs. *Hess, A.* .. 91
Induced Movements in Newborn and Very Young Rats, Both Normal and Spinal Animals. *Roofe, P. G.* 117
The Regeneration of Peripheral Nerves Inserted into the Cerebral Cortex and the Healing of Cerebral Lesions. *Clemente, C. D.* 123

Isolated Lesions of the Fastigial Nuclei in the Cat

Attempts were made to produce localized lesions of the fastigial nuclei in a series of 30 cats in order to study the resulting physiologic deficits and the associated fiber degeneration. Lesions of the fastigial nuclei were produced unilaterally in 15 cats and bilaterally in 5 others. The following conclusions were drawn:

1. Unilateral lesions of the fastigial nucleus produce bilateral degeneration in the uncinate fasciculus, which is quantitatively more abundant on the side opposite the lesion.
2. Unilateral lesions of the fastigial nucleus produce ipsilateral fiber degeneration in the justarestiform body which terminates in the vestibular nuclei, particularly Deiters' nucleus.
3. Fibers of the descending limb of the uncinate fasciculus project to all vestibular nuclei, though the greatest number of fibers pass to the lateral and spinal vestibular nuclei, and to the dorsal paramedian area of the reticular formation of the pons and medulla.
4. Fibers of the ascending limb of the uncinate fasciculus pass rostrally dorsal to the brachium conjunctivum and at the level of the inferior colliculus the fibers begin to disperse.

Fibers are projected to the nuclei of the posterior commissure, the posterior commissure, the nuclei of the lateral lemniscus and at thalamic levels enter the parafascicular and centromedian nuclei.

5. Fibers originating from the fastigial nuclei do not ascend in the brachium conjunctivum and do not enter or terminate in the red nuclei, the motor nuclei of any of the cranial nerves, the medial longitudinal fasciculus or any of the so-called cerebellar relay nuclei.

6. Unilateral lesions of the fastigial nucleus in the cat produce disturbances of equilibrium and ataxia involving axial and appendicular musculature; these disturbances undergo relatively rapid attenuation according to a specific pattern.

7. Physiologic disturbances resulting from complete and incomplete unilateral destruction of the fastigial nuclei could not be distinguished by the nature or severity of the deficits.

8. Bilateral lesions of the fastigial nuclei in the cat provoke quantitatively more severe and enduring ataxia and disturbances of equilibrium than unilateral lesions, and the attenuation is prolonged.

Hypotheses presented are:

1. Physiologic phenomena resulting from localized destruction of the fastigial nuclei are probably due to the removal of inhibitory influences of this structure upon the vestibular nuclei.

2. The ascending limb of the uncinate fasciculus may relay vestibular impulses to thalamic and other supra segmental levels.

(Authors' Abstr.)

VOL. 50 J. COMP. PHYSIOL. PSYCHOL. OCTOBER, 1957

The Chick's "Preference" for Some Visual Properties of Water. *Rheingold, H. L., and Hess, E. H.* .. 417
Form Preferences in Newly Hatched Chicks. *Fantz, R. L.* .. 422
Saccharine Reinforcement in a T Maze. *Hughes, L. H.* .. 431
Differentiation Learning by Monkeys. *McClearn, G. E.* .. 436
Solution of Bent-Wire Problems by Monkeys and Chimpanzees. *Davis, R. T., et al.* .. 441
The Gradient of Delay of Secondary Reward in Avoidance Learning. *Kamin, L. J.* .. 445
The Gradient of Delay of Secondary Reward in Avoidance Learning Tested on Avoidance Trials Only. *Kamin, L. J.* .. 450
The Retention of an Incompletely Learned Avoidance Response. *Kamin, L. J.* .. 457
Some Effects of Response-Independent Positive Reinforcement on Maintained Operant Behavior. *Herrnstein, R. J., and Morse, W. H.* .. 461
Discrimination of Intermittent Photic Stimulation in the Rat Without Its Striate Cortex. *Schwartz, A. S., and Clark, G.* .. 468
Olfactory Discrimination Deficits in Monkeys with Temporal Lobe Ablations. *Santibañez, G., and Pinto Hamuy, T.* .. 472
Sexual Behavior in Adult Male Hamsters Castrated-Adrenalectomized Prior to Puberty. *Warren, R. P., and Aronson, L. R.* .. 475
Thermal Behavior of the Subnourished and Pantothenic-Acid-Deprived Rat. *Weiss, B.* 481
Random and Food-Directed Activity in Hyperphagic and Normal Rats. *Teitelbaum, P.* 486
The Effect of Activity Restriction upon Adjustment to Cyclic Food Deprivation. *Reid, L. S., and Finger, F. W.* .. 491
The Effect of Reinforcement upon Activity During Cyclic Food Deprivation. *Finger, F. W., et al.* .. 495
Relative Acceptability of Sodium Chloride and Sucrose Solutions. *Young, P. T., and Asdourian, D.* .. 499
The Relative Strengths of Consummatory Responses in Hunger, Thirst, and Exploratory Drive. *Zimbardo, P. G., and Montgomery, K. C.* .. 504
Exploratory Behavior in Two Mazes with Dissimilar Alternatives. *Williams, C. D., and Kuchta, J. C.* .. 509
Response by Rats to Differential Stimulus Complexity. *Dember, W. N., et al.* .. 514
An Exploration of Abdominal Potentials. *Davis, R. C., et al.* .. 519
Continuous Recording of Arterial Pressure: An Analysis of the Problem. *Davis, R. C.* 524
Effects of Infantile and Adult Shock-Trauma upon Learning in the Adult White Rat. *Baron, A., et al.* .. 530
The Role of Pre-injury Education and Intelligence Level in Intellectual Loss After Brain Injury. *Weinstein, S., and Teuber, H.-L.* .. 535

DECEMBER

Traumatic Avoidance Learning: The Effects of Conditioned Stimulus Length in a Free-Responding Situation. *Brush, E. S.* .. 541
The Effects of Shock Intensity on the Acquisition and Extinction of an Avoidance Response in Dogs. *Brush, F. R.* .. 547
Maintenance of Avoidance Behavior by Unavoidable Shocks. *Sidman, M., et al.* .. 553
The Use of Shock-Contingent Variations in Response-Shock Intervals for the Maintenance of Avoidance Behavior. *Sidman, M., and Boren, J. J.* .. 558
Expectancy and the Extinction of Expectancy in the Rat. *Goodson, F. E., et al.* .. 563
The Role of the Sequential Cue in Behavior Fixation. *Feldman, R. S., and Waite, R. R.* 567

Conditioned Reinforcement in Chimpanzees. *Kelleher, R. T.* 571
Direct Manipulation of the Relevant Cue and Difficulty of Discrimination. *Schrier, A. M., and Harlow, H. F.* 576
The Effect of Shuffled Pairs on the Learning of Serial Discrimination Problems by Monkeys. *Leary, R. W.* 581
Multiple Discrimination Learning in Raccoons. *Shell, W. F., and Riopelle, A. J.* 585
Learning in a Multiple-Door Situation Under Various Drive States. *Jerome, E. A., et al.* 588
A Test of the Discrepancy Hypothesis of Motivation Using Intensity of Visual Stimulation. *Lubow, R. E., and Tighe, T. J.* 592
Activity in the White Rat as a Function of Apparatus and Hunger. *Strong, P. N., Jr.* 596
Some Physiological Factors that Regulate Eating Behavior. *Smith, M., and Duffy, M.* 601
Infantile Experience and Consummatory Behavior in Adulthood. *Levine, S.* 609
The Effect of Handling at Various Age Levels on Emotional Behavior of Adult Rats. *Gertz, B.* 613
Experimental Analysis of the Hoarding Habit in the Rat: IV. Terminal Reinforcement Followed by High Drive at Test. *Marx, M. H., and Brownstein, A. J.* 617
Exploratory Behavior of Three Strains of Rats. *Carr, R. M., and Williams, C. D.* 621
The Effect of Bilateral Hippocampal Stimulation on the Acquisition and Extinction of an Instrumental Response. *Correll, R. E.* 624
Effects of Circumscribed Cortical Lesions upon Somesthetic and Visual Discrimination in the Monkey. *Wilson, M.* 630
Performance of Visual Discriminations Presented Tachistoscopically in Monkeys with Temporal Neocortical Ablations. *Chow, K. L., and Orbach, J.* 636
Effects of Electroshock Convulsions upon the Learning Performance of Monkeys: IV. Discrimination-Reversal Learning. *Braun, H. W., et al.* 641
The Effect of ECS on Retention in Young and Adult Rats. *Thompson, R.* 644
Physiological Costs Incurred by Dextro-Amphetamine. *Hauty, G. T., et al.* 647

VOL. 51 FEBRUARY, 1958

Long-Term Light Adaptation of the Human Electroretinogram. *Armington, J. C., and Biersdorf, W. R.* 1
Positive Reinforcement Induced by Intracerebral Stimulation in the Monkey. *Bursten, B., and Delgado, J. M. R.* 6
Drinking in the Rat Induced by Hypertonic Saline. *Wayner, M. J., Jr., and Reimanis, G.* 11
Water Regulation in the Rat: I. Gastrointestinal Exchange Rates of Water and Sodium Chloride in Thirsty Animals. *O'Kelly, L. I., et al.* 16
Water Regulation in the Rat: II. The Effects of Preloads of Water and Sodium Chloride on the Bar-Pressing Performance of Thirsty Rats. *O'Kelly, L. I., and Falk, J. L.* 22
Pentobarbital Facilitation of Water Ingestion in the Albino Rat. *Schmidt, H., Jr.* 26
Chlorpromazine Depression of Food Intake in the Albino Rat. *Schmidt, H., Jr., and Van Meter, W. G.* 29
Induction of Broodiness by Participation in Courtship and Nest-Building in the Ring Dove (*Streptopelia Risoria*). *Lehrman, D. S.* 32
Normal Sexual Behavior in Male Rats Isolated at Fourteen Days of Age. *Beach, F. A.* 37
Learning in Kittens with Manipulatory, Exploratory, and Food Incentives. *Miles, R. C.* 39
Facilitation of Exploration by Hunger in Rats. *Zimbardo, P. G., and Miller, N. E.* 43
Intensity of Illumination and the Rate of Responding in a Multiple-Door Situation. *Jerome, E. A., et al.* 47
Learning as a Function of Change of Sensory Stimulation in Food-Deprived and Food Satiated Animals. *Forgays, D. G., and Levin, H.* 50
Determinants of Emotionality in the Rat: III. Strain Differences. *Broadhurst, P. L.* 55
Learning Factors in Social Facilitation and Social Inhibition in Rats. *Holder, E. E.* 60
Effects of Delayed Reward on Choice Behavior and Response Speeds in Children. *Lipsitt, L. P., and Castaneda, A.* 65
Formation of Conditioned Responses in Infant Monkeys. *Mason, W. A., and Harlow, H. F.* 68
Performance of Infant Rhesus Monkeys on a Spatial Discrimination Problem. *Mason, W. A., and Harlow, H. F.* 71
The Effect of Different Kinds of Form Pre-Exposure on Form Discrimination Learning. *Forgus, R. H.* 75
Object-Quality Discrimination Learning Sets in Mental Defectives. *Ellis, N. R.* 79
Analysis of Serial-Discrimination Learning by Monkeys. *Leary, R. W.* 82
Effects of Reinforcing Single Stimuli upon Subsequent Discrimination Learning by Rhesus Monkeys. *Sinha, M. M.* 87
The Question of Insight and Delayed Reaction in Fish. *Munn, N. L.* 92
Spatial Perception in the Hooded Rat: Cue Dominance and Direct Orientation. *Shaw, W. A., and Seidel, R. J.* 98
An Experimental Study of the Relationship Between Secondary Reinforcing and Cue Effects of a Stimulus. *Wyckoff, L. B., et al.* 103
Retention in *Paramecium Aurelia*. *Gelber, B.* 110

Effect of Delay Between Training and Extinction on the Extinction of an Avoidance Response. *Moyer, K. E.* 116

The Effect of Varying the Intertrial Interval in Discrimination Learning by Normal and Brain-Operated Monkeys. *Riopelle, A. J., and Churukian, G. A.* 119

Differential Effects of Temporal Neo-Cortical Resections on Overtrained and Non-Overtrained Visual Habits in Monkeys. *Orbach, J., and Fantz, R. L.* 126

Effects of Rhinencephalic Lesions on Conditioning of Avoidance Responses in the Rat. *Thomas, G. J., and Otis, L. S.* 130

APRIL

Ingestion Patterns in Hyperphagic and Normal Rats. *Teitelbaum, P., and Campbell, B. A.* 135

Effect of Female Sex Hormones on Incubation Behavior in the Ring Dove (*Streptopelia Risoria*). *Lehrman, D. S.* 142

Effects of Hydration on the Running and Drinking Performance of Thirsty Rats. *Solarz, A. K.* 146

Color Vision in the Marmoset. *Miles, R. C.* 152

A Methodological Study of "Shape Constancy" in the Rhesus Monkey. *Zeigler, H. P., and Leibowitz, H.* 155

Effects of Rhinencephalic Lesions on Maze Learning in Rats. *Thomas, G. J., and Otis, L. S.* 161

The Effect of Ventral Rhinencephalic Lesions on Avoidance Thresholds in Monkeys. *Weiskrantz, L., and Wilson, W. A., Jr.* 167

Effect of Sensory Stimulation on the Activity of Normal and Prefrontal-Lobectomized Monkeys. *Isaac, W., and DeVito, J. L.* 172

The Effects of Cranial X-Radiation on Maze Acquisition in Rats. *Blair, W. C.* 175

Maze Learning in Pre- and Neonatally X-Irradiated Rats. *Furchtgott, E., et al.* 178

The Effect of Whole-Body X-Irradiation on the Electroconvulsive Threshold in the Rat. *Schwartzbaum, J. S., et al.* 181

Electrical and Behavioral Effects of Different Types of Shock Stimuli on the Rat. *Campbell, B. A., and Teghtsoonian, R.* 185

The Relationship Between Stimulation Voltage and Rate of Hypothalamic Self-Stimulation in the Rat. *Reynolds, R. W.* 193

Shock Motivated Serial Discrimination Learning and the Chemical Block of Autonomic Impulses. *Arbit, J.* 199

The Effect of Anoxia on the Acquisition of a Discrimination Habit. *Bryant, J. H., and Thompson, R.* 202

Inhalation of Carbon Dioxide Has No Effect on Conditioned Emotional Responses in Rats. *Otis, L. S., and Thomas, G. J.* 205

Effects of Chlorpromazine and Electroconvulsive Shock on a Conditioned Emotional Response. *Heistad, G. T.* 209

Learning and Hormone Activity: I. Effects of Thyroid Levels Upon the Acquisition and Extinction of an Operant Response. *Denenberg, V. H., and Myers, R. D.* 213

Effects of Amobarbital Sodium and Dexamphetamine Sulfate on the Conditioning of the Eyeblink Response. *Franks, C. M., and Trouton, D.* 220

Conditioning of Skin Temperature Changes in Dogs. *Chambers, R. M., and Fuller, J. L.* 223

Integrated Muscle Action Potentials in a Weight-Lifting Task as a Function of Weight and Rate of Lifting. *Small, A. M., Jr., and Gross, N. B.* 227

Noxious Stimulation in Infant and Adult Rats and Consummatory Behavior. *Levine, S.* 230

Imprinting: The Interaction of Learned and Innate Behavior: III. Practice Effects on Performance, Retention, and Fear. *Jaynes, J.* 234

Imprinting: The Interaction of Learned and Innate Behavior: IV. Generalization and Emergent Discrimination. *Jaynes, J.* 238

Apparent Learning in the Paramecium. *Katz, M. S., and Deterline, W. A.* 243

Rate of Responding as a Function of Intertrial Interval. *Jerome, E. A., et al.* 248

Partial Reinforcement and Nonresponse Acquisition. *Lewis, D. J., and Cotton, J. W.* 251

The Effect of a Food-Associated Stimulus on Operant-Level Locomotor Behavior. *Gilbert, T. F., and Sturdivant, E. R.* 255

The Effect of an Irrelevant Motive on Learning. *Miles, R. C.* 258

JUNE

Conditioned Reflexes and Leão's Spreading Cortical Depression. *Bures, J., et al.* 263

Roughness Discrimination After Penetrating Brain Injury in Man: Analysis According to Locus of Lesion. *Weinstein, S., et al.* 269

Effects of Delaying Reward on Visual-Discrimination Performance in Monkeys with Frontal Lesions. *Mishkin, M., and Weiskrantz, L.* 276

The Role of the Suprapharyngeal Ganglion in Spontaneous Alternation and Negative Movements in *Lumbricus Terrestris L. Wayner, M. J., Jr., and Zellner, D. K.* 282

The Effect of Ophthalmectomy upon Responses of the Rat to Radiation and Taste Stimuli. *Garcia, J., and Kimeldorf, D. J.* 288

Locomotor Coordination Following Pre- and Neonatal X Irradiation. *Furchtgott, E., and Echols, M.* 292

Effects of Local Blood Removal on Skin Resistance and Potential. *Wilcott, R. C.* .. 295
Conditioning of a Hyperinsulin Type of Behavior in the White Rat. *Reiss, W. J.* .. 301
Studies on Sugar Preference: I. The Preference for Glucose Solutions and its Modification by Injections of Insulin. *Jacobs, H. L.* .. 304
Learning and Hormone Activity: II. Effects of Thyroid Levels upon Retention of an Operant Response and upon Performance Under Starvation. *Denenberg, V. H., and Myers, R. D.* .. 311
The Interresponse Interval as a Measure of Bar-Pressing Behavior in Normal and Drugged Rats. *Stone, G. C., et al.* .. 315
Effects of Hunger and Male Sex Hormone on Self-Stimulation of the Brain. *Olds, J.* 320
Aftereffects of Copulatory Activity of the Male Rat: I. *Larsson, K.* .. 325
Color Vision in the Squirrel Monkey. *Miles, R. C.* .. 328
Stimulus Satiation in Situations Without Choice. *Glanzer, M.* .. 332
Running Behavior as a Function of Apparatus and of Restriction of Goal Box Activity. *Cotton, J. W., et al.* .. 336
Hoarding in Hamsters with Systematically Controlled Pretest Experience. *Bevan, W., and Grodsky, M. A.* .. 342
Secondary Reinforcement as a Function of the Amount of Primary Reinforcement. *Butter, C. M., and Thomas, D. R.* .. 346
A Replication and Further Analysis of a Study on Position Reversal Learning in Hungry and Thirsty Rats. *Bolles, R. C.* .. 349
Response Strength as a Function of Hours of Food Deprivation Under a Controlled Maintenance Schedule. *Birch, D., et al.* .. 350
The Effects of Various Punishment-Escape Events upon Subsequent Choice Behavior of Rats. *Buchanan, G. N.* .. 355
The Effects of Irregular Learning Conditions upon the Rate and Permanence of Learning. *McNamara, H. J., and Wike, E. L.* .. 363
The Effect of Different Amounts of Training on the Resistance to Extinction of Different Patterns of Partially Reinforced Responses. *Capaldi, E. J.* .. 367
The Effect of Differential Training upon the Relative Strength of Place vs. Response Habits. *Aderman, M.* .. 372
Learning Sets and Object-Size Effects in Visual Discrimination Learning by Raccoons. *Johnson, J. I., Jr., and Michels, K. M.* .. 376
Avoidance Conditioning as a Factor in the Effects of Unavoidable Shocks on Food-Reinforced Behavior. *Herrnstein, R. J., and Sidman, M.* .. 380
Transposition of Response to Size in Children. *Rudel, R. G.* .. 386

AUGUST

Rapid Escape Learning Without Avoidance Learning Motivated by Hypothalamic Stimulation in Cats. *Roberts, W. W.* .. 391
Both Rewarding and Punishing Effects from Stimulation of Posterior Hypothalamus of Cat with Same Electrode at Same Intensity. *Roberts, W. W.* .. 400
Analysis of Simple Searching Behavior in the Frontal Monkey. *Meyer, D. R., and Settlage, P. H.* .. 408
Temporal Determinants of Foveal CFF After Lesions of the Cerebral Visual Pathways. *Battersby, W. S., and Bender, M. B.* .. 411
Aftereffects of Copulatory Activity of the Male Rat: II. *Larsson, K.* .. 417
The Effect of Intracranial Stimulation on Memory in Cats. *Thompson, R.* .. 421
Visual and Tactual Discriminations by Macaques with Serial Temporal and Parietal Lesions. *Pasik, P., et al.* .. 427
Effects of Subcortical Lesions in Monkeys on Visual-Discrimination and Single-Alternation Performance. *Rosvold, H. E., et al.* .. 437
Thyroid Hormone and Conditioning. *Richards, W. J., and Stockburger, J. C.* .. 445
An Effect of the Vital Dye Trypan Red on Approach-Avoidance Conflict. *Poschel, B. P. H.* .. 448
Visual Discrimination of the Orientation of Rectangles by *Octopus Vulgaris* Lamarck. *Sutherland, N. S.* .. 452
The Learning of Incidental Cues by Rhesus Monkeys. *Brown, W. L., and Carr, R. M.* 459
The Facilitative Effect of Sensory Preconditioning on Instrumental Learning in the Rhesus Monkey. *Brown, W. L., et al.* .. 461
Discrimination After Pretraining on Component Stimuli. *Gentry, G. V., et al.* .. 464
Progressive Discrimination Learning in Platyrrhine Monkeys. *Shell, W. F., and Riopelle, A. J.* .. 467
The Learning of Detours and Barriers by Monkeys. *Davis, R. T.* .. 471
Latency of the Conditioned Heart Rate as a Function of the CS-US Interval. *Church, R. M., and Black, A. H.* .. 478
Further Experiments on Prolonged Exposure to Visual Forms: The Effect of Single Stimuli and Prior Reinforcement. *Walk, R. D., et al.* .. 483
The Relationship Between Age and Avoidance Learning in the Hooded Rat. *Denenberg, V. H., and Kline, N. J.* .. 488

Correction vs. Noncorrection with Changing Reinforcement Schedules. *Parducci, A., and Polt, J.* 492
The Reinforcing Effect of Weak-Light Onset as a Function of Amount of Food Deprivation. *Davis, J. D.* 496
The Acquisition and Extinction of an Instrumental Response Under Constant and Variable Stimulus Conditions. *Brown, J. S., and Bass, B.* 499
The Effect of Effort upon Extinction. *Capehart, J., et al.* 505
Generalization of Fixated Behavior in the Rat. *Ellen, P., and Feldman, R. S.* 508
Effect of Early Food Deprivation on Adult Behavior in the Rat. *Mandler, J. M.* 513

VOL. 54 J. EX. PSYCHOL. OCTOBER, 1957

Probabilistic Discrimination Learning. *Estes, W. K., et al.* 233
Statistical Learning Theory Applied to an Instrumental Avoidance Situation. *Brody, A. L.* 240
The Length-Difficulty Relationship in Compound Trial-and-Error Learning. *Noble, C. E.* 246
Effect of Pattern Variation Upon Verbal Maze Learning. *Thompson, R. F., et al.* 253
Incidental Learning at Five Stages of Intentional Learning. *Bahrick, H. P.* 259
Effects of Force and Amplitude Cues on Learning and Performance in a Complex Tracking Task. *Briggs, G. E., et al.* 262
Effect of Variation in Associative Frequency of Stimulus and Response Members on Paired-Associate Learning. *Mandler, G., and Campbell, E. H.* 269
Studies of Distributed Practice: XVII. Interlist Interference and the Retention of Paired Consonant Syllables. *Underwood, B. J., and Richardson, J.* 274
Secondary Extinction of Lever-Pressing Behavior in the Albino Rat. *Rozeboom, W. W.* 280
Error Reinforcement in a Modified Serial Perceptual-Motor Task. *Marx, M. H., and* 288 *Goldbeck, R. A.* 288
Conditioning of Muscle Action Potential Responses Resulting From Passive Hand Movement. *Doehring, D. G.* 292
Problem Solving in Multiple-Goal Situations. *Anderson, S. B.* 297

NOVEMBER

A Test of a Statistical Learning Theory Model for Two-Choice Behavior With Double Stimulus Events. *Anderson, N. H., and Grant, D. A.* 305
Reward and Punishment in a Minimal Social Situation. *Sidowski, J. B.* 318
Effects of Varied Distance on Short-Range Size Judgments. *Jenkin, N.* 327
The Interaction of Association Value and Stimulus Configuration in Size Estimation. *Dow, M. E., and Gordon, J. E.* 332
Semantic Generalization With Experimentally Induced Associations. *Jeffrey, W. E., and Kaplan, R. J.* 336
The Immediate Effects of Emotionality Upon Behavior Strength. *Ellis, N. R.* 339
Emotionality and the Yerkes-Dodson Law. *Broadhurst, P. L.* 345
Transfer From Verbal Pretraining to Motor Performance as a Function of Response Similarity and Angle of Movement. *Hoffeld, D. R.* 353
The Discrimination of Speech Sounds Within and Across Phoneme Boundaries. *Liberman, A. M., et al.* 358
Retention of Verbal Associates as a Function of Strength. *Runquist, W. N.* 369
Supplementary Report: Effect of Verbal Pretraining on the Acquisition of a Complex Motor Skill. *Battig, W. F., et al.* 375

DECEMBER

Ratio Scales and Category Scales for a Dozen Perceptual Continua. *Stevens, S. S., and Galanter, E. H.* 377
Intelligibility as a Function of Frequency of Usage. *Rosenzweig, M. R., and Postman, L.* 412
Selective Sampling in Discrimination Learning. *LaBerge, D. L., and Smith, A.* 423
The Semantic Differential and Mediated Generalization as Measures of Meaning. *Lipton, L., and Blanton, R. L.* 431
Cutaneous Discrimination of Radiant Heat. *Teichner, W. H.* 438
The Role of Prefeeding in an Apparent Frustration Effect. *Seward, J. P., et al.* 445
Resistance to Extinction as a Function of the Discrimination Habit Established During Fixed-Ratio Reinforcement. *Denny, M. R., et al.* 451
Judgments of Visual Velocity as a Function of Length of Observation Time. *Goldstein, A. G.* 457
Effect of Amount of Interpolated Learning and Time Interval Before Test on Retention in Rats. *Frankmann, J. P.* 462
Classical Conditioning of Human Pupillary Dilation. *Gerall, A. A., et al.* 467

VOL. 55 JANUARY, 1958

On the Use of Inconsistency of Preferences in Psychological Measurement. *Coombs, C. H.* 1
Methodological Aspects of Auditory Threshold Measurements. *Corso, J. F., and Cohen, A.* 8

An Analysis of Positioning Movements and Static Reactions. *Fleishman, E. A.*	13
Age Differences in Transfer and Retroaction as a Function of Intertask Response Similarity. *Gladis, M., and Braun, H. W.*	25
A Goalless Gradient. *Pereboom, A. C.*	31
Two-Category Judgments of Sequences of Stimuli of Two Values. *DeSoto, C.*	34
Factors in Individual Improvement in Solving Twenty-Questions Problems. *Faust, W. L.*	39
Effect of Brightness of Simultaneous Visual Stimulation on Absolute Auditory Sensitivity. *Thompson, R. F., et al.*	45
Differential Conditioning and Intensity of the UCS. *Runquist, W. N., et al.*	51
Mediated Verbal Similarity as a Determinant of the Generalization of a Conditioned GSR. *Phillips, L. W.*	56
Transfer After Training With Single Versus Multiple Tasks. *Duncan, C. P.*	63
Effect of a Warning Signal Preceding a Noxious Stimulus on Verbal Rate and Heart Rate. *Kanfer, F. H.*	73
Utility of Grades: Level of Aspiration in a Decision Theory Context. *Becker, S. W., and Siegel, S.*	81
The Empirical Validity of Equal Discriminability Scaling. *Alluisi, E. A., and Sidorsky, R. C.*	86
Supplementary Report: Interlist Interference and the Retention of Paired Consonant Syllables. *Underwood, B. J., and Richardson, J.*	95

FEBRUARY

Studies of Pupillary Conditioning. *Young, F. A.*	97
"Backward" Associations in Transfer and Learning. *Murdock, B. B., Jr.*	111
Probability Learning Under Equivalent Data Collection Methods. *Komorita, S. S.*	115
Expectation and Resistance to Extinction of a Lever-Pulling Response as a Function of Percentage of Reinforcement and Number of Acquisition Trials. *Lewis, D. J., and Duncan, C. P.*	121
Effects of Mediated Associations on Transfer in Paired-Associate Learning. *Norcross, K. J., and Spiker, C. C.*	129
Transfer Effects From a Single to a Double Integral Tracking System. *Briggs, G. E., et al.*	135
Some Informational Aspects of Form Discrimination. *Krulee, G. K.*	143
Assimilation and Contrast Effects of Anchoring Stimuli on Judgments. *Sherif, M., et al.*	150
Achievement Drive and Habitual Modes of Task Approach as Factors in Skill Transfer. *Miles, G. H.*	156
Influence of Task Complexity and Instructions Upon Simple and Discrimination Reaction Times. *Sidowski, J. B., et al.*	163
Effects of Task Difficulty, Stimulus Similarity, and Type of Response on Stimulus Predifferentiation. *Murdock, B. B., Jr.*	167
Motor Conflict Behavior as a Function of Motivation and Amount of Training. *Andreas, B. G.*	173
Perceptual Efficiency as Related to Induced Muscular Effort and Manifest Anxiety. *Shore, M. F.*	179
Evidence That the Secondary Reinforcing Stimulus Must be Discriminated. *McGuigan, F. J., and Crockett, F.*	184
Muscle Responses and Their Relation to Rote Learning. *Berry, R. N., and Davis, R. C.*	188
Cue and Contextual Stimulus Intensity in Discrimination Learning. *Nygaard, J. E.*	195
Shifts in Connotative Meaning of Words as a Function of Previous Restrictive Experience. *Mayzner, M. S., and Tresselt, M. E.*	200

MARCH

Inferential Behavior in Children: I. The Influence of Reinforcement and Incentive Motivation. *Kendler, H. H., et al.*	207
Pre-Extinction in Sensory Preconditioning. *Coppock, W. J.*	213
Context Effects With Judgmental Language That is Absolute, Extensive, and Extra-Experimentally Anchored. *Campbell, D. T., et al.*	220
Time Sharing Between Frequency-Coded Auditory and Visual Channels. *Klemmer, E. T.*	229
A Correlational Study of Two Reasoning Problems. *Brunk, L., et al.*	236
Eyelid Conditioning as a Function of Unconditioned Stimulus Intensity and Intertrial Interval. *Prokasy, W. F., Jr., et al.*	242
Verbal and Motor Responses to Seven Symbolic Visual Codes: A Study in S-R Compatibility. *Alluisi, E. A., and Muller, P. F., Jr.*	247
"Permanent" Satiation Phenomena With Kinesthetic Figural Aftereffects. *Wertheimer, M., and Leventhal, C. M.*	255
Transfer to a Motor Task as Influenced by Conditions and Degree of Prior Discrimination Training. *Goss, A. E., and Greenfeld, N.*	258
Retention in Motor Learning as a Function of Amount of Practice and Rest. *Jahnke, J. C.*	270
Acquisition and Extinction of Problem-Solving Set. *Gardner, R. A., and Runquist, W. N.*	274

Concept Formation as a Function of Competition Between Response Produced Cues. *Kendler, H. H., and Karasik, A. D.*	278
Time-Order Errors in Comparative Judgments of Hurtfulness. *Geertsma, R. H.*	284
The Influence of Complexity and Novelty in Visual Figures on Orienting Responses. *Berlyne, D. E.*	289
Interaction Between Total Stimulus Information and Specific Stimulus Information in Visual Recognition. *Newbrough, J. R.*	297
Supplementary Report: Effect of Addition of Irrelevant Verbal Cues on Perceptual-Motor Learning. *Braun, H. W., and Bendig, A. W.*	301

APRIL

Marginal and Conditional Stimulus and Response Probabilities in Verbal Conditioning. *Engler, J.*	303
Long-Term Retention of Perceptual-Motor Skills. *Ammons, R. B., et al.*	318
Meaning, Frequency, and Visual Duration Threshold. *Taylor, J. A.*	329
Acquisition and Extinction of the Conditioned Eyelid Response Following Partial and Continuous Reinforcement. *Reynolds, W. F.*	335
An Extension of Hullian Theory to Response Decrements Resulting From Drugs. *Payne, R. B.*	342
Conditioned-Stimulus Variables in Avoidance Learning. *Schwartz, M.*	347
Age Differences in Short-Term Retention of Rapidly Changing Information. *Kirchner, W. K.*	352
A New Technique for Studying Spatial Generalization With Voluntary Responses. *Brown, J. S., et al.*	359
The Effects of Delay of Reinforcement Upon an Operant Discrimination in the Pigeon. *Myers, J. L.*	363
An Analysis of Two-Person Game Situations in Terms of Statistical Learning Theory. *Atkinson, R. C., and Suppes, P.*	369
Variable Frequency of Knowledge of Results and the Learning of a Simple Skill. *Bilodeau, E. A., and I. McD.*	379
Information Processing and Problem Solving. *Solley, C. M., and Snyder, F. W.*	384
Distance Constancy. *Smith, O. W.*	388
Apparent Backward Association: A Situational Effect. *Storms, L. H.*	390

MAY

Partial and 100% Reinforcement Under Chance and Skill Conditions. *James, W. H., and Rotter, J. B.*	397
UCS Intensity and the Associative (Habit) Strength of the Eyelid CR. *Spence, K. W., et al.*	404
A Further Test of the Hypothesis of Autonomous Memory Trace Change. *Lovibond, S. H.*	412
Effects of Window Size and Two Conditions of Judgment on the Reproduction of Objective Velocity. *Smith, O. W.*	416
The Dependence of Probability of Response on Size of Step Interval in the Method of Limits. *Brackmann, J., and Collier, G.*	423
Nonindependence of Successive Responses at the Visual Threshold as a Function of Interpolated Stimuli. *Collier, G., and Verplanck, W. S.*	429
Dimensional Analysis of Movement Reactions. *Fleishman, E. A.*	438
The Dimensional Analysis of a New Series of Facial Expressions. *Engen, T., et al.*	454
Task-Influence and the Stability of Generalized Expectancies. *Rychlak, J. F.*	459
Ease of Attainment of Concepts as a Function of Response Dominance Variance. *Freedman, J. L., and Mednick, S. A.*	463
Isolation Effects: Stimulus and Response Generalization as Explanatory Concepts. *Newman, S. E., and Saltz, E.*	467
Effects of Strength of Drive on Learning and on Extinction. *Barry, H., III*	473
Half-Loudness Judgments Without Prior Stimulus Context. *Garner, W. R.*	482
Age Changes and Information Loss in Performance of a Pursuit Tracking Task Involving Interrupted Preview. *Griew, S.*	486
Effects of Previously Associated Annoying Stimuli (Auditory) on Visual Recognition Thresholds. *Hochberg, J., and Brooks, V.*	490
Position Preference and Discrimination Learning. *Goer, M. H.*	492
The Effects of Degree of Learning and Problem Difficulty on Perseveration. *Thompson, R.*	496
Conditioning of the Human Pupillary Dilation Response as a Function of the CS-UCS Interval. *Gerall, A. A., and Woodward, J. K.*	501

JUNE

Stimulus and Response Generalization: Tests of a Model Relating Generalization to Distance in Psychological Space. *Shepard, R. N.*	509
Acquisition and Change of a Concept Attitude as a Function of Consistency of Reinforcement. *Rhine, R. J., and Silun, B. A.*	524

The Relation of Visual Acuity to Convergence and Accommodation. *Tulving, E.* .. 530
Reproductive Interference Following "Appropriate" and "Inappropriate" Warm-Up Activities. *Thune, L. E.* 535
The Relation of Eye Movements, Body Motility, and External Stimuli to Dream Content. *Dement, W., and Wolpert, E. A.* 543
Associative Interference in Verbal Paired-Associate Learning. *Besch, N. F., and Reynolds, W. F.* 554
Effects of Stress and Anxiety on Performance of a Complex Verbal-Coding Task. *Katchmar, L. T., et al.* 559
Mediated Stimulus Equivalence and Distinctiveness in Human Conditioning. *Grice, G. R., and Davis, J. D.* 565
Serial Verbal Learning Under Two Conditions of Hunger Motivation. *Lerner, R. G., et al.* 572
Achievement Motivation and Goal-Setting Behavior on a Learning Task. *Kausler, D. H., and Trapp, E. P.* 575
Effect of Knowledge of Conditioning Upon Eyelid Conditioning. *McAllister, W. R., and D. E.* 579
Habit Reversal as a Function of Schedule of Reinforcement and Drive Strength. *Kendler, H. H., and Lachman, R.* 584
Comparing Reversal and Nonreversal Shifts in Concept Formation With Partial Reinforcement Controlled. *Harrow, M., and Friedman, G. B.* 592
Summation of Manifest Anxiety and Muscular Tension. *Meyer, D. R., and Noble, M. E.* 599
Variation of Temporal Intervals Among Critical Events in Five Studies of Knowledge of Results. *Bilodeau, E. A., and I. McD.* 603
Temporal Effects of Conditioned Fear on the Eyelid Reflex. *Spence, K. W., and Runquist, W. N.* 613
Binaural Supplementation of the Auditory Intensive Threshold. *Shaw, W. A.* .. 617
Progressive Ambiguity in the Attainment of Concepts on the Wisconsin Card Sorting Test. *Gormezano, I., and Grant, D. A.* 621
Sequential Decision Making: Wald's Model and Estimates of Parameters. *Becker, G. M.* 628
The Relationship Between Discriminability and Generalization: A Re-Evaluation. *Kalish, H. I.* 637

VOL. 56 JULY

Ready Signals and the Effect of Interpolated UCS Presentations in Eyelid Conditioning. *Dufort, R. H., and Kimble, G. A.* 1
Differential Effects of "Display Lags" and "Control Lags" on the Performance of Manual Tracking Systems. *Garvey, W. D., et al.* 8
Influence of Intertrial Interval During Extinction on Spontaneous Recovery of Conditioned Eyelid Responses. *Howat, M. G., and Grant, D. A.* 11
Independence in the Learning of Two Consecutive Responses Per Trial. *Brody, A. L.* 16
Discrimination Learning in a Verbal Conditioning Situation. *Popper, J., and Atkinson, R. C.* 21
Effect of the Spatial Relationship Between Cue, Reward, and Response in Simple Discrimination Learning. *Murphy, J. V., and Miller, R. E.* 26
Delay of Reinforcement, Response Perseveration, and Discrimination Reversal. *Pubols, B. H., Jr.* 32
Discrimination Learning as a Function of Pretraining Reinforcement Schedules. *Stevenson, H. W., and Pirojnikoff, L. A.* 41
A Positive Correlation Between Action Decrement and Learning. *Walker, E. L., and Paradise, N.* 45
Amount and Percentage of Reinforcement and Duration of Goal Confinement in Conditioning and Extinction. *Hulse, S. H., Jr.* 48
Complex Incidental Learning as a Function of Anxiety and Task Difficulty. *Spielberger, C. D., et al.* 58
Associative Connection Between Paired Verbal Items. *Richardson, J., and Erlebacher, A.* 62
Changes in Distribution of Muscular Tension During Psychomotor Performance. *Gregg, L. W.* 70
A Relationship Between Incentive Motivation and Ability Level in Psychomotor Performance. *Fleishman, E. A.* 78
Instrumental and Competing Behavior as a Function of Trials and Reward Magnitude. *Pereboom, A. C., and Crawford, B. M.* 82
Studies in Incidental Learning: VII. Effects of Frequency of Exercise and Length of List. *Postman, L., and Adams, P. A.* 86
Supplementary Report: Discrimination Learning in Rats as a Function of Highly Distributed Trials. *Pennington, D. F., and Thompson, R.* 94
Replication Report: Two- and Three-Choice Verbal-Conditioning Phenomena. *Cotton, J. W., and Rechtschaffen, A.* 96

AUGUST

Effects of Exposure Time and Intertrial Interval Upon Decrement to the Müller-Lyer Illusion. *Mountjoy, P. T.* 97
Human Trial-and-Error Learning Under Joint Variation of Locus of Reward and Type of Pacing. *Noble, C. E., and J. L.* 103
Conditioning and Hypnosis. *Das, J. P.* 110
Stimulus Similarity and Sequence of Stimulus Presentation in Paired-Associate Learning. *Rothkopf, E. Z.* 114
Associative Transfer in Motor Paired-Associate Learning as a Function of Amount of First-Task Practice. *Spiker, C. C., and Holton, R. B.* 123
Recognition of Novel Visual Configurations With and Without Eye Movements. *Mooney, C. M.* 133
Verbal Conditioning and Extinction With Verbal and Nonverbal Reinforcers. *Buss, A. H., et al.* 139
Meaningfulness of Material, Distribution of Practice, and Serial-Position Curves. *Braun, H. W., and Heymann, S. P.* 146
Acquisition and Extinction of a Partially Reinforced Running Response at a 24-Hour Intertrial Interval. *Weinstock, S.* 151
Effects of Shock-Induced Stress on Verbal Performance. *Chiles, W. D.* 159
Anxiety Level and Ego Involvement as Factors in Concept Formation. *Romanow, C. V.* 166
R-S Learning as a Function of Meaningfulness and Degree of S-R Learning. *Jantz, E. M., and Underwood, B. J.* 174
Comment on "Role of Prefeeding in an Apparent Frustration Effect". *Amsel, A.* .. 180
Supplementary Report: Stability of a Verbal Rate Change in Experimental Anxiety. *Kanfer, F. H.* 182
Supplementary Report: Complexity and Orienting Responses With Longer Exposures. *Berlyne, D. E.* 183

SEPTEMBER

Probability Learning in Children. *Stevenson, H. W., and Zigler, E. F.* 185
The Interaction of Ability and Amount of Practice With Stimulus and Response Meaningfulness (m, m^1) in Paired-Associate Learning. *Cieutat, V. J., et al.* .. 193
Some Variables Affecting Latent Extinction. *Thomas, A. R.* 203
Studies of Distributed Practice: XVIII. The Influence of Meaningfulness and Intralist Similarity of Serial Nonsense Lists. *Underwood, B. J., and Richardson, J.* .. 213
An Investigation of Mediation in Preconditioning. *Seidel, R. J.* 220
A Comparison Between Rote Learning and Concept Formation. *Metzger, R.* .. 226
The Interaction of Pitch and Loudness Discriminations. *Harris, J. D., et al.* 232
The Effects of Response Contingent Probabilities Which Favor Response Change. *Shelly, M. W., II* 239
Stimulus Spacing and the Judgment of Loudness. *Stevens, J. C.* 246
Resistance to Extinction as a Function of Continuous or Intermittent Presentation of a Training Cue. *Marx, M. H.* 251
The Effect of Intertrial Responding on Conditioning and Extinction of Avoidance Behavior. *Murphy, J. V., and Miller, R. E.* 256
The Recognition, Naming, and Reconstruction of Visual Figures as a Function of Contour Redundancy. *Anderson, N. S., and Leonard, J. A.* 262
Body Temperature and Psychological Ratings During Sleep Deprivation. *Murray, E. J., et al.* 271
Deprivation and Generalization. *Jenkins, W. O., et al.* 274
Gradients in Response Percentages as Indices of Nonspatial Generalization. *Bass, B.* 278
Effects of Previous Experience and Information on Performance on a Word-Formation Problem. *Battig, W. F.* 282
Time Sharing as an Index of Automatization. *Bahrick, H. P., and Shelly, C.* 288
The Temporal Factor in Reward and Nonreward of Monkeys. *Leary, R. W.* 294

VOL. 58 J. GEN. PSYCHOL. JANUARY, 1958

The Value of Anticipated Events as a Determinant of Probability Learning and Extinction. *Crandall, V. J., et al.* 3
Critical Flicker Frequency in Light- and Dark-Adaptation. *Dondero, A., et al.* .. 11
Place-Learning, Cognitive Maps, and Parsimony. *Birch, H. G., and Korn, S. J.* .. 17
*Exceptional Memory for Dates and Weather in a Schizoid Psychopath. *Robertson, J. P. S.* 37
Personality Rigidity as Measured by Aniseikonic Lenses and by Perceptual Tests of Metabolic Efficiency. *Wertheimer, M., and Aronson, E.* 41
Behavior as Performance: A Note on Muenzinger's Frame of Reference for the Study of Behavior. *Walker, K. F.* 51
The Organization of the Retention of Verbal Material. *Brand, H., and Woods, P. J.* 55
Goal Setting Rigidity as a Function of Anxiety and Task-Ambiguity. *Himelstein, P.* 69
Perceptual Defense: An Integration with Other Research Findings. *Chodorkoff, B., and J.* 75

The Completeness and Accuracy of Counseling Interview Reports. *Froehlich, C. P.* 81
A Note on Estimating the Statistical Significance of Mutuality. *Katz, L., et al.* .. 97
A Gestalt Law of Mental Work. *O'Connell, D. C.* 105
Anxiety as a Factor Influencing Routine Performance Under Auditory Stimuli. *Auble, D., and Britton, N.* 111
Some Suggestions for the Conceptual and Theoretical Analysis of Complex Intervening Variables in Problem-Solving Behavior. *Marx, M. H.* 115

Exceptional Memory for Dates and Weather in a Schizoid Psychopath

The patient was a laboratory technician aged 27. The chief of the unusual skills he possessed was that given any date in the preceding 16 years he could rapidly say on what day of the week it fell and what the weather was. He had lost a succession of jobs and repeatedly failed examinations. Therefore he had consulted an occupational psychologist who found that in most abilities he was above the 90th percentile of the professional class. Because of the oddities in his behaviour he was advised to seek neuropsychiatric help. The neuropsychiatrist to whom he went was particularly interested in his memory ability and after his admission as an in-patient referred him to the psychological department for elucidation of this. The principal abnormal features of the patient's behaviour were numerous stereotyped mannerisms in gestures and movements, exaggeratedly deliberate enunciation of speech, and a tendency to express himself in cliches wherever possible. There was no evidence of thought disorder, delusions, or hallucinations.

In the psychological session the patient was asked the weekday of 80 dates presented in randomized order but systematically covering the 16-year period. He was right in 74 and the other six were wrong by a day only. He showed strong self-involvement with his skill which made searching consideration of his method of working difficult. It emerged, however, that he operated by a number of anchor dates of which he knew the weekday and related all other dates to these by invariant calendar relations. From further investigation it was possible to identify 118 anchor dates. These all related to either personal events or unusual weather. The total number at his disposal was estimated as more than 500. His memory for weather was more troublesome to check precisely but in 12 presented instances his statement corresponded to the contemporary newspaper account. In connection with each anchor date he recollected the sequence of weather conditions up to the next anchor date. In fact dates, weather, and personal events were linked in an intricate system of cross-association.

He had certain other memory skills. He knew all squares up to that of 1,000 and all cubes up to that of 100. He could multiply four-place by four-place numbers in his head. His account indicated that he worked by factorization with auditory-motor imagery. He also experienced synesthesia, associating a constant colour image with each number from 0 to 9, but this was not utilized in his calculations. In all tests of abilities applied to him his scores were very high except in the Hanfmann-Kasanin where he showed marked concreteness of thinking. While answering questions in the various parts of the examination but particularly in calculations his muscles became very tense and he exhibited the mannerisms of rapidly twirling and untwirling a handkerchief. Biographical inquiry indicated that he had been fond of studying calendars and almanacs from an early age. The development of his special skills, however, began at the age of 11 after hearing a lightning calculator on the radio. It was then that he started a diary, recording important personal events and the weather and committing special dates to memory. He disclaimed all knowledge of the lives and methods of the classic arithmetical prodigies.

The various tests and enquiries suggested that his skills could be analysed as dependent on the following factors: (*a*) a capacity to remember discrete details much above average, (*b*) high numerical ability, (*c*) a special interest in numbers, regular sequences, and recurrences, and weather, (*d*) a narrow range of general interests, (*e*) unusual concern with the minor events of his own life, (*f*) fondness for reviewing his life with or without the aid of diaries, (*g*) a habit of relating each event as it occurred to its date, weekday, and weather, (*h*) increasing self-involvement with his skills and deliberate cultivation of a large reserve of anchor dates, and (*i*) incessant practice.

The patient was treated with insulin and left the hospital after a few months. He has remained without neuropsychiatric care for over eight years now, showing neither improvement nor deterioration of symptoms, as a recent follow-up revealed. The pattern of losing jobs and failing examinations continued, his mannerisms have remained unchanged. His parents, who are quite well-off, are tolerant of his peculiarities. His memory skill continues as the chief interest of his life. Not only has he maintained it but he has tried to make it cover an earlier period than his diaries, taking national events as anchor dates and learning the weather from past records.

(Author's Abstr.)

APRIL

The Development of Factor Analysis. *Royce, J. R.* 139
Relative Resistance to Extinction of Aperiodic and Continuous Reinforcement Separately and in Combination. *Likely, F. A.* 165
The Concept of Motivation. *Bunch, M. E.* 189
The Private Practice of Psychotherapy: A Clinical Psychologist's Report. *Ellis, A.* .. 207

Expressed Attitudes and Needs of Religious Persons Compared with Those Determined by Projective Techniques. *Dreger, R. M.* .. 217
A Psychometric Study of Institutionalized Epileptics on the Wechsler-Bellevue. *Angers, W. P.* .. 225
The Stratification of Words in Cognitive Organization. *Benedetti, D. T.* .. 249
Subskills in Rotary Pursuit as Affected by Rate and Accuracy Requirements and by Distribution of Practice. *Ammons, R. B., et al.* .. 259
Judged Intelligence: An Attempt to Apply the Psychophysical Approach to a Traditionally Psychometric Problem-Area. *Bevan, W., and Bansavage, J. W.* .. 281
Phonetic Symbolism with Respect to Three Dimensions from the Semantic Differential. *Birch, D., and Erickson, M.* .. 291

VOL. 59 JULY, 1958

A Factor Analysis of Two Foreign Language Aptitude Batteries. *Carroll, J. B.* .. 3
Idiographic Knowledge. *O'Connell, D. C.* .. 21
Rational Psychotherapy. *Ellis, A.* .. 35
Objective Measures of Needs and Course Achievement in Introductory Psychology. *Bendig, A. W.* .. 51
Potential Intelligence: A Learning Theory Approach to Description and Clinical Implication. *Mahrer, A. R.* .. 59
A New Level of Aspiration Test and An Application of It. *Gruen, A.* .. 73
Satiation and the Rate of Lapse of Verbal Meaning. *Wertheimer, M., and Gillis, W. M.* 79
The Effects of Anxiety and Experimentally-Induced Stress on Verbal Learning. *Kalish, H. I., et al.* .. 87
The Temporal Gradient in *GSR* Conditioning. *Bierbaum, W. B.* .. 97
The Effect of Thiamine on Thiamine Sniff Thresholds. *Comrey, A. L., et al.* .. 105
Relationship Between Visual and Auditory Discrimination and Anxiety Level. *Jones, O.* 111
Single Standard Judgment: A Reorientation in Rating Scale Procedure. *Webb, S. C., and Bevan, W.* .. 119
Spontaneous Recovery of the Conditioned Eyelid Response. *Grant, D. A., et al.* .. 135

OCTOBER

Effect of Reserpine on Avoidance of Humans by Rhesus Monkeys. *Schrier, A. M., and Harlow, H. F.* .. 149
A Comparison of Figural After-Effects from the Perception of Real and of Apparent Movement. *Train, L. E., and Walthall, W. J., Jr.* .. 157
Music Therapy for the Mentally Ill: I. A Historical Sketch and a Brief Review of the Literature on the Physiological Effects and on Analysis of the Elements of Music. *Paul, R., and Staudt, V. M.* .. 167
Delay and Risk: Influence of a Delay in Performance on Maximum Risk-Taking Levels. *Dearnaley, E. J.* .. 177
The Significance of Assumptive and Philosophic Operations in Psychological Methodology. *Newbury, E.* .. 185
Secondary Reinforcement and Avoidance Conditioning. *Murphy, J. V., et al.* .. 201
The Effects of Emotionality on Concept Formation. *Sweetland, A., and Childs-Quay, L.* 211
The Minnesota Teacher Attitude Inventory in an American Minority-Group School Setting: II. Indirect Validation as a Test of Pupil-Directedness. *Fishman, J. A.* 219
A Historical Note on Retention. *Meissner, W. W.* .. 229
Progression from Simple to Complex as a Molar Law of Learning. *Campbell, D. T., and Gruen, W.* .. 237
Hypnotherapy with Borderline Schizophrenics. *Ellis, A.* .. 245
Serial Position Effects and the "Marbe Effect" in the Free Recall of Meaningful Words. *Bousfield, W. A., et al.* .. 255
Randomized Stimuli and the Non-Independence of Successive Responses at the Visual Threshold. *Verplanck, W. S., and Blough, D. S.* .. 263
An Experimental Investigation of "Non-Randomness" in Parapsychological Targets and Calls. *Smith, K., and Curran, S. A.* .. 273

VOL. 92 J. GENET. PSYCHOL. MARCH, 1958

Psychotherapy with Adolescent Girls in a Court Clinic. *Sherman, M. H.* .. 3
Effect of Adrenalectomy on Anxiety Motivated Behavior. *Moyer, K. E.* .. 11
Effect of Adrenalectomy on Emotional Elimination. *Moyer, K. E.* .. 17
A Formula for Selecting Toys for Nondirective Play Therapy. *Lebo, D.* .. 23
Hunger Motivation in the Rat Following Local Application of Cobalt-60. *Shaklee, A. B., and Miller, W. C.* .. 35
An Analysis of Manifest Content of the Earliest Memories of Children. *Weiland, I. H., and Steisel, I. M.* .. 41
Latent Changes in the Food Preferences of Irradiated Monkeys. *Davis, R. T.* .. 53
Rosenzweig Picture-Frustration Study Results with Minority Group Juvenile Delinquents. *Norman, R. D., and Kleinfeld, G. J.* .. 61
Parents' Attitudes and Adjustment of Children. *Burchinal, L. G.* .. 69

Marginal Social Values of Rural School Children. *Witryol, S. L., and Calkins, J. E.* 81
A Study of Developmental Changes in Conceptual and Perceptual Associative Clustering. *Bousfield, W. A., et al.* 95
Mothers' and Fathers' Differences in Parental Acceptance of Children for Controlled Comparisons Based on Parental and Family Characteristics. *Burchinal, L. G.* 103

JUNE

The Influence of Learning in Activity Wheel Behavior. *Hall, J. F.* 121
Insight vs. Conditioning. *Hulicka, I. M., and Johnson, B. G.* 127
The Expressed Values of Institutionalized Delinquent Boys. *Trent, R. D.* 133
Children's Personality Adjustment and the Socio-economic Status of Their Families. *Burchinal, L., et al.* 149
Parental Judgment of Responsibility in Children, and Children's Adjustment. *Harris, D. B.* 161
Maternal Attitudes Toward Child Behavior and Their Relationship to the Diagnostic Category of the Child. *Abbe, A. E.* 167
Adrenal Changes Paralleling Short-Range Behavioral Effects of Electroshock. *Shaklee, A. B., et al.* 175
Audiogenic Seizures in Rats After Gonadectomy and During Subsequent Replacement Therapy. *Bevan, W., et al.* 183
Changes in Ethnocentrism Associated with a Two-Year College Experience. *Plant, W. T.* 189
Attitude Toward Aging of Individuals with Experiences with the Aged. *Tuckman, J., and Lorge, I.* 199
The Plasticity of the Physique from Early-Adolescence Through Adulthood. *Zuk, G. H.* 205
Studies in Social Perception: Methodology. *Estvan, F. J.* 215
Effect of Injected Adrenalin on an Avoidance Response in the Rat. *Moyer, K. E., and Bunnell, B. N.* 247

VOL. 93 SEPTEMBER

The Relationship Between Incidence of Specific Gastro-Intestinal Reactions of the Infant and Psychological Characteristics of the Mother. *Pinneau, S. R., and Hopper, H. E.* 3
Sex-Appropriate Behavior in Adolescence. *Zuk, G. H.* 15
Effects of Satiation on Food Preferences in Monkeys. *Warren, J. M.* 33
The Performance of X-ray Irradiated and Nonirradiated Rhesus Monkeys Before, During, and Following Chronic Barbiturate Sedation. *Davis, R. T., et al.* 37
Sex, Intelligence, and Sorority or Fraternity Membership and Changes in Ethnocentrism Over a Two-Year Period. *Plant, W. T.* 53
Chlorpromazine and Habit Reversal. *Shaklee, A. B.* 59
Comparisons of Distractibility in Irradiated and Nonirradiated Monkeys. *McDowell. A. A.* 63
Facilitative Effects of Irradiation on Performance of Monkeys on Discrimination Problems with Reduced Stimulus Cues. *McDowell, A. A., and Brown, W. L.* 73
Reactions of Superior and Feebleminded Children to an Illusion. *Dixon, J. C.* 79
A Study of Socialization Patterns at the High School Level. *Jones, M. C.* 87
Some Psychological Factors in the Reading of Fiction. *Thayer, L. O., and Pronko, N. H.* 113
The Relationship Between Children's Play Interests and Their Mental Ability. *Wang, J. D.* 119
An Analysis of Expressions Used by Adolescents at Varying Economic Levels to Describe Accepted and Rejected Peers. *Feinberg, M. R., et al.* 133
Stereotyped Associations to Schematic Faces. *Nash, H.* 149
A Study of the Needs Status of Adolescents from Broken Homes. *Bartlett, C. J., and Horrocks, J. E.* 153

VOL. 14 J. INDIVID. PSYCHOL. MAY, 1958

The Place of the Individual and of Idiodynamics in Psychology: a Dialogue. *Rosenzweig, S.* 3
Assumptions in Psychology. *van Kaam, A. L.* 22
Four Phenomenologies. *Landsman, T.* 29
Existential Psychoanalysis and Individual Psychology. *Stern, A.* 38
Emotional Blocks to Creativity. *Maslow, A. H.* 51
Suicide. *Adler, A.* 57
Experimental Studies of Organ Inferiority. *Overton, R. K.* 62
Attitudes Towards One's Name and One's Self. *Strunk, O., Jr.* 64
Life Style in Schizophrenia. *Adler, K. A.* 68
Adlerian Concepts of Schizophrenia: Symposium. *Adler, A., and Bierer, J.* 73
Reflections on Ernest Jones' Biography of Freud. *Bottome, P.* 79

VOL. 2 J. MENT. DEFIC. RES. JUNE, 1958

The Significance of Twins in Mongolism in the Light of New Evidence. *Keay, A. J.* 1
Phenylketonuria: The Phenylalanine-Tyrosine Ratio in the Detection of the Heterozygous Carrier. *Hsia, D. Y.-Y.* 8

Tranquillizers in Mental Deficiency: Meprobamate. *Craft, M.*	17
The Rote and Concept Learning of Imbeciles. *Hermelin, B., and O'Connor, N.*	21
The Influence of a Retarded Child Upon Family Limitation. *Holt, K. S.*	28

VOL. 125 **J. NERV. MENT. DIS.** APRIL-JUNE, 1957

Psychiatric Disorders Associated with Childbirth. *Ostwald, P. F., and Regan, P. F., III*	153
*Aromatic Metabolism in Schizophrenia. I. Statistical Evidence for Aromaturia. *McGeer, P. L., et al.*	166
*Aromatic Metabolism in Schizophrenia. II. Bidimensional Urinary Chromatograms. *McGeer, E. G., et al.*	176
The Psychosomatic Concept of Obesity. *Kaplan, H. I., and H. S.*	181
An Example of the Psychopathology of Religion. The Seventh-day Adventist Denomination. *Yarnell, H.*	202
Convulsions in Multiple Sclerosis. *Feldman, S.*	213
*Impairment of Abstracting Ability in Multiple Sclerosis. *Parsons, O. A., et al.*	221
Schizophrenia and Cortical Involvement. *Walker, R. G.*	226
The Potentiating Effect of Diphenhydramine Hydrochloride (Benadryl). *Rosner, S.*	229
Some Factors in Chronic Ward Recovery. *O'Reilly, P. O.*	232
Recreational Counseling in the Psychiatric Service of a General Hospital. *Olson, W. E., and McCormack, J. B.*	237
Some Physiological Responses in Neurotics. *Ferguson, R. S.*	240
The Structure of Emotion. II. The Physical Basis of Anxiety. *Stanley-Jones, D.*	247
The Structure of Emotion, III. The Physiology of the Oedipus Complex. *Stanley-Jones, D.*	259
Brief Chlorpromazine Treatment of Disturbed Patients in a Psychiatric Reception Hospital. *Deutsch, L., et al.*	273
A Psychiatrist Looks at Epilepsy. *Power, T. D.*	279
Commitment of the Mentally Ill: "Treatment" or Social Restraint? *Szasz, T. S.*	293
Hallucination: A Problem in Neurophysiology. *Levin, M.*	308
The Effect of Group Administration upon Symptom Formation under LSD. *Slater, P. E., et al.*	312
Psychopharmacological Studies on Suppression. *Clark, L. D., and Beecher, H. K.*	316
The Effect of Divergent Viewpoints in Analytical Psychotherapy Training. *Sager, C. J.*	322
Working with the Stuttering Problem. *Barbara, D. A.*	329
*A Comparison of Monoureide and Diureide Sedatives. With Respect to Drug Habituation, Untoward Effects and Mortality Rates. *Feinblatt, T. M., et al.*	335
Xanthoma of the Choroid Plexus in a Child. *Rosner, S.*	339

Aromatic Metabolism in Schizophrenia. I. Statistical Evidence for Aromaturia

1. The work of 20 authors who have reported on aromatemia or aromaturia in schizophrenia has been briefly reviewed.

2. The overnight urine of 109 schizophrenics, 315 non-schizophrenic mental patients and 223 normals has been studied chromatographically. The schizophrenics appeared to excrete a higher than normal concentration of aromatic compounds. Based on an arbitrary scale which takes into account the number of coloured areas and the intensity of these areas on an unidimensional chromatogram, the schizophrenics have a rating of 8·1 ±0·3, the non-schizophrenics 5·0±0·2, and the normals 5·0±0·1.

3. The chromatographic rating has been found to vary from day to day for both schizophrenic and normal individuals.

4. The distribution curves of the normal, non-schizophrenic and schizophrenic groups show considerable overlap: this overlap is attributed to: (*a*) daily variation in the aromatic excretion, (*b*) diagnostic error, (*c*) error in measurement, and (*d*) unknown factors.

5. Diet and emotional stress are not considered to play a dominant role in this aromaturia.

(Authors' Abstr.)

Aromatic Metabolism in Schizophrenia. II. Bidimensional Urinary Chromatograms

The significance of the comparative studies reported is that they serve to confirm previous quantitative reports of excessive aromatic excretion by schizophrenics. They indicate the possibility that a qualitative difference may exist as well.

(Authors' Abstr.)

Impairment of Abstracting Ability in Multiple Sclerosis

In view of experimental and clinical findings it is predicted that patients with multiple sclerosis will manifest a loss in the ability to function at the abstract level. Seventeen M.S. patients, 14 males and 3 females, are closely matched with 17 control patients having no C.N.S. disorder on age, sex, education, intelligence, and race. Both groups are given the Wechsler-Bellevue Intelligence Scale, Form I, and the Grassi Block Substitution Test, the latter requiring different levels of functioning on the abstract-concrete continuum. The results confirm the prediction: the M.S. patients perform significantly poorer on the G.B.S.T., especially on the abstract levels. Additional analysis controlling for the possible interfering effects of visual-motor difficulty in M.S. gives rise to the same results. It is concluded that a moderate loss of the ability to abstract in patients with multiple sclerosis is demonstrated and that such a loss roughly parallels extent of neurological disturbance.

(Authors' Abstr.)

A Comparison of Monoureide and Diureide Sedatives. With Respect to Drug Habituation, Untoward Effects and Mortality Rates

1. Three separate clinical investigations, including a total of 371 case studies and a comprehensive survey of medical literature, failed to show a single case or manifestation of drug habituation following continued use of acetylcarbromal or bromisovalum (bromural), singly or in combination.

2. The absence of untoward effects attributed to acetylcarbromal or bromisovalum is in sharp contrast to the frequent and serious neurological and psychiatric manifestations reported and the common occurrence of drug habituation following use of the barbiturates.

3. Due to the fact that the monoureide sedatives are non-habit-forming, have no side effects, no hangover and no contraindications, the authors believe that the acetylcarbromal bromisovalum combination is much preferable for continued use as a sedative or somnifacient to the dangerous and habit-forming barbiturates.

(Authors' Abstr.)

VOL. 126 JANUARY, 1958

A Cataract of Consequences. *Gregg, A.* 3
*The Nature of Apraxia. *Denny-Brown, D.* 9
Some Aspects of Amino Acid and Protein Metabolism of the Nervous System. *Waelsch, H.* 33
*Visceral Brain, Its Component Parts and Their Connections. *Papez, J. W.* 40
*Effects of Septal and Amygdaloid Lesions on Emotional Behavior and Conditioned Avoidance Responses in the Rat. *King, F. A.* 57
The Severely Defective Individual. *Sarason, S. B., and Gladwin, T.* 64
On the Participation of Adaptive Hormones in Disease Susceptibility. *Selye, H.* .. 97

The Nature of Apraxia

It is concluded that in its highest elaboration mimetic and propositional behaviour is related to conceptual organization of space, objects and persons, in the dominant hemisphere. Such behaviour is more highly vulnerable to pathologic changes than is direct physiologic reaction to external events. The resulting disorder is ideational apraxia. Perseveration of identification is a mild form of the same defect. Acquired specialized motor function that requires a leading hand for its performance is closely related to cortical dominance but can be affected independently of symbolic function. The relationship is probably through the conceptual defect underlying constructional apraxia. Related symbolism and dexterity may be separately managed by different hemispheres, independently vulnerable to unilateral lesion, and independently controlling the subcortical motor apparatus for the two sides.

Apraxia that is independent of conceptual function (kinetic apraxia) can be traced to the abnormal operation of released sensory factors that direct the physiological aspects of behaviour.

There are two general types of disturbance, exemplified by frontal and parietal kinetic apraxia, both of which show perseveration of sensory effects. Bilateral kinetic apraxia first and most seriously affects gait and other bilateral motor performance. When the normal equilibrium of cortical praxis is already disturbed, it is possible to induce tremor by concurrent stimuli with conflicting effect. Subcortical extra-pyramidal lesions release the same apractic reactions in persistent (dystonic) form. There are more commonly mixed forms where conflicting reflex effects lead to instability of posture, athetosis and tremor. The static (tonic) aspects of such disorders of movement can be identified with the prolonged operation (perseveration) of sensory factors.

(Authors' Abstr.)

Visceral Brain, its Component Parts and Their Connections

The olfactory, visceral, hippocampal and hypothalamic neural mechanisms are described. Their interconnections receive special attention. Together they form the visceral brain or limbic lobe. Working together these several parts subserve the basic biologic needs of the individual. Olfaction takes part in primitive search for food, feeding and identification of odorous objects in the environment. A primitive olfactory "motor" pathway from the nucleus basalis to the habenula, thence to interpeduncular nucleus, to tegmental and reticular formation is illustrated. The visceral cortex, claustrum, uncinate bundle and amygdala are described with some interpretation. Their relations to the original olfactory mechanisms, internal capsule, and basal ganglia are indicated. The hippocampal formation, as a cortical dependency, and its mechanism for integration of emotional states, arousal to action, hypothalamic and cortical responses are considered. Hippocampal, neuronal mechanism is illustrated. The hypothalamic nuclei, their amygdalar, forebrain, hippocampal, autonomic and hypophysial connections are illustrated and defined. It is pointed out that large-celled nuclei conduct nerve impulses; whereas the granule cell nuclei may possibly have a cholinergic function. Most of the reactions of the visceral brain are biologically grounded, close to the reflex-like level of innate activities. They are designed to preserve, maintain and protect the individual, and control his vegetative needs, an important segment of inner life. From this point of view each mechanism appears to have an appropriate structural design.

(Author's Abstr.)

Effects of Septal and Amygdaloid Lesions on Emotional Behaviour and Conditioned Avoidance Responses in the Rat

A study was conducted with rats to investigate the effects of septal and amygdaloid lesions upon avoidance conditioning, and overt emotional behaviour. The latter was measured on a six-component rating scale. Conditioning was carried out in a shuttle box, so arranged that the rat could move through a door from one compartment to another to avoid a shock signalled by a buzzer. Results were as follows:

1. Septal lesions produced a dramatic increase in overt emotionality, whereas unoperated controls and operated controls with neocortical and cingulate lesions showed no such damage.
2. Septal lesions decreased the number of trials required to learn the avoidance response.
3. Lesions in the amygdaloid nuclei of normal animals did not significantly alter overt emotionality.
4. There were no differences in rate of acquisition of conditioned responses between normal rats and rats with amygdaloid damage. However, the response latencies of the amygdaloid preparations were significantly lengthened. Throughout the experiment, no other group differed from normals in response latency.

(Author's Abstr.)

FEBRUARY

Psychotherapeutics and the Schizophrenic Reaction. *Will, O. A., Jr.* 109
Neurophysiologic Studies of Personality. *Shagass, C., and Kerenyi, A. B.* 141
*The Cingulates and Behavior. *Pechtel, C., et al.* 148
Pattern Stability and Correlates of Photic-Electroencephalographic Activation. *Ulett, G. A., and Johnson, L. C.* 153
*Glutamic Oxalacetic Transaminase in Cerebrospinal Fluid. *Miyazaki, M.* 169
*Clinical and Laboratory Observations on LSD-25. *Feld, M., et al.* 176
*Some Pharmacologic Correlations of the Chemotherapy of Mental Disease. *Truitt, E. B., Jr.* 184

The Cingulates and Behaviour

Nine cats and 11 monkeys were studied for from 3 to 12 months preoperatively for (*a*) spontaneous behaviour, (*b*) learning capacity, and (*c*) the neurotogenic effects of motivational conflicts. Ablation of cingulate areas 23 and 24 then produced the following results:

1. Minimal amnesia for previous learning, but increased incidence of error and impaired precision of acquiring new skills.
2. A tendency to diffuse and precipitate activity.
3. Diminished motivation, especially for initiating or completing goal directed tasks, with the exception of area 24 monkeys.
4. Moderately increased aggressivity toward other animals and man.
5. Lowered thresholds of startle and fear, except in three cynamologos monkeys.
6. An accentuation of the effects of isolation in exacerbating the above reactions.
7. In one instance, maternal pre-emption of food at the expense of a nursing baby.
8. Minimal or no relief of phobic, regressive, socially maladaptive, and other aberrant behaviour in the neurotic animals, but with greater amenability to retraining and other therapy.
9. Generally increased susceptibility to the reinduction of experimental neuroses, again with the exception that two area 24 monkeys showed an increased tolerance to conflictual experiences and in one of these the authors were unable to induce a neurosis even after 6 months of effort.

The theoretical and clinical significance of these findings is briefly discussed.

(Authors' Abstr.)

Glutamic Oxalacetic Transaminase in Cerebrospinal Fluid

1. The control value of glutamic oxalacetic transaminase of the spinal fluid among 30 subjects was $16 \cdot 8 \pm 7 \cdot 6$ units.
2. A significant elevation of spinal fluid GOT in acute cerebrovascular accidents was noted in 9 out of 32 tested patients, comprising 28 per cent. of the total.
3. In patients with acute cerebrovascular accidents high spinal fluid GOT levels tend to indicate poor prognosis and low GOT levels a favourable prognosis.
4. The spinal fluid GOT in patients with brain tumours was normal except in those with obstructive hydrocephalus. In one case of congenital internal hydrocephalus there was marked elevation.
5. The convulsive disorders showed elevated spinal fluid GOT only when the specimens were taken during or soon after the end of the convulsion.

6. The spinal fluid GOT was found to be normal in the other neurologic diseases included in this series.

7. Transaminase levels in one human brain was highest in the cerebellum, followed by the pons, cerebral cortex and white matter, hypothalamus and thalamus, in that order.

(Author's Abstr.)

Clinical and Laboratory Observations on LSD-25

1. LSD-25 can be used to demonstrate the integration of basic neurophysiology with well-known concepts of psychopathology.

2. LSD-25 has the astonishing quality of bringing into focus the patient's repressed emotional attitudes, conflicts, etc., and permits their reactivation. The hallucinatory experiences are not always symbolic, but in the majority of the patients these were not difficult to interpret with knowledge of the patient's developmental history, defences, etc.

3. The intense transference develops and may be capitalized on with direct, interpretive, Rosen type psychotherapy centering on the re-experiences as well as for reconditioning of "breaking up" of obsessiveness circuits in favour of more appropriate thinking circuits. Improvement was also produced by psychocatharsis.

4. LSD-25 effects are mediated through the reticular activating substance and hypothalamic-pituitary-adrenal axis, as demonstrated physiologically.

5. In the number of cases presented, most had placebos for control and all except the chronic brain syndromes demonstrated some degree of improvement in the sense that they were able to return to society and their families in a more productive capacity.

(Authors' Abstr.)

Some Pharmacologic Correlations of the Chemotherapy of Mental Disease

In concluding the review of the dynamically active field of the pharmacotherapy of mental disease one is tempted to point out three generalizations of drug action which appear to be crystallizing.

1. Mental depression may be related to either a neurohumoral deficiency of cerebral serotonin or some sympathomimetic amine or to some enzyme disorder such as a monoamine oxidase over-activity in the adrenergic excitatory mechanisms of the brain which might produce such a deficiency.

2. Drugs which are most effective in curbing the acute mania, motor excitement and hallucinations of the schizophrenic reaction are those which interfere in some manner with the adrenergic excitatory centres for emotional responses in the brain as exemplified by reserpine and chlorpromazine.

3. Psychoneurotic symptoms respond most favourably to drugs which have a minimum of side effects and produce a relaxation of neuromuscular tension and anxiety with a minimum of drowsiness and other symptoms of central nervous system depression.

(Author's Abstr.)

MARCH

Biochemical Aspects of Schizophrenia. *Hoagland, H.*	211
Statistical Control Studies in Neurology. IX. The Gordon Sign. *Madonick, M. J.* ..	221
Early Object Relations, Somatic, Affective, and Personal. *Greene, W. A., Jr.* ..	225
Electroencephalographic Findings in Aged Psychiatric Patients. *Obrist, W. D., and Henry, C. E.*	254
The Effect of Meprobamate on the Electroencephalogram. *Henry, C. E., and Obrist, W. D.*	268
*The Effects of Two Antiserotonin Substances (BAS and BAB) and of Bufotenin on the Spontaneous Electric Activity of the Brain. *Rinaldi, F.*	272
*Clinical Evaluation of BAS (Benzyl Analog of Serotonin): A Tranquilizing Drug. *Rudy, L. H., et al.*	284
*Studies on the 5-Hydroxytryptamine (Serotonin) Content in Human Brain. *Costa, E., and Aprison, M. H.*	289
Exchange Transfusions in Schizophrenic Patients. *Freedman, A. M., and Ginsberg, V.*	294

The Effects of Two Antiserotonin Substances (BAS and BAB) and of Bufotenin on the Spontaneous Electric Activity of the Brain

1. BAS (benzyldimethylserotonin) induces convulsive-like non-spreading electric discharges in the hippocampus of the rabbit.

2. BAB (benzyldimethylbufotenin) produces continuous fast activity localized to the lenticular nucleus, chiefly in the putamen. The amplitude is three to four times larger than that of waves of comparable frequency normally present in the lenticular nucleus and in other parts of the rabbit brain.

3. Bufotenin (N,N-dimethylserotonin) causes the disappearance of slow wave high voltage activity and introduces an enduring "alert" desynchronized pattern.

4. The electroencephalogram in 12 to 15 chronic psychotic patients did not show significant changes under treatment with BAS. In three subjects only, however, a decrease of the amplitude and slowing of the fundamental alpha activity were observed.

(Author's Abstr.)

Clinical Evaluation of BAS (Benzyl Analog of Serotonin): A Tranquilizing Drug

The effects of BAS, the benzyl analog of serotonin, were studied on 24 chronic, moderately disturbed female psychotic patients. The most prominent beneficial action, that of tranquilization, was noted in 22 of these patients. Tranquilization was attained at dosage levels varying between 50 to 200 mg. per day. The maximum tolerated dose was 300 mg. per day. Among the paranoid patients who improved, the delusions became less important. The intensity and frequency of the hallucinatory experiences diminished. In hebephrenic patients, speech became less dissociated. These salutary psychiatric effects, however, were marred by untoward reactions involving the general health of some patients who lost weight chiefly because of a profound feeling of weakness and fatigue. Another disturbing side reaction deserving comment is ataxia which was extensive in some patients. The effects of BAS are compared with those of reserpine which they resemble in many ways, but are not seen to the same extent with both drugs. The clinical similarities of BAS and reserpine are ascribed to the indole group, which their chemical structures have in common. Their different non-indolic moieties are regarded as being the causes for the clinical dissimilarities exhibited by these two drugs.

(Authors' Abstr.)

Studies on the 5-Hydroxytryptamine (Serotonin) Content in Human Brain

The presence of serotonin in human brain structures has been demonstrated. The areas of the allocortex had higher serotonin concentrations than the isocortex. The highest concentration of serotonin per unit weight of tissue is in the mesencephalic structures (mainly substantia nigra and probably red nucleus); the diencephalic structures (hypothalamus) contain also relatively large amounts of this neurohormone. The cerebellum as well as the isocortex have the lowest concentrations of serotonin.

(Authors' Abstr.)

APRIL

The Development of Concepts Relating to the Electrical Activity of the Brain. *Brazier, M. A. B.* 303
The Psychologic Importance of the Uterus and Its Functions. *Drellich, M. G., and Bieber, I.* 322
Somato-Psychotherapeutic Approach to Long Term Schizophrenic Patients: Three Year Evaluation. *Shatin, L.* 337
Freud and Existentialism. *Tiebout, H. M., Jr.* 341
*Psychologic Changes Induced by Reserpine Therapy on a Group of Severely Disturbed Psychotics. *Tanck, R. H.* 353
Electric Shock Casualties. *Raskin, N.* 360
The Life and Activity of I. M. Sechenov. *Ischlondsky, N.* 367
Electroencephalographic Finding During Chlorpromazine-Diethazine Treatment. *Denber, H. C. B.* 392

Psychologic Changes Induced by Reserpine Therapy on a Group of Severely Disturbed Psychotics

Eighty-eight psychiatric patients, nearly all schizophrenic and all considered to be among the most severely disturbed, were divided into matched pairs to form an experimental (reserpine) and a control group (placebo). A preliminary evaluation was made using Lorr's Multidimensional Scale for Rating Psychiatric Patients, an abbreviated Wechsler Bellevue Intelligence Scale, and individual Rorschach. The experimental group then received reserpine and the control group placebo for a period of 7 weeks. Then a second evaluation was made. The medications were reversed and a third measure was made 7 weeks after the switch in medications.

Results show that the average patient of this study becomes more compliant, submissive, communicative, and outgoing, but perhaps somewhat more dysphoric after reserpine therapy. Intellectual functioning as shown by Wechsler-Bellevue performance is improved, but there seems to be no significant improvement in potential ego strength as shown by the application of Klopfer's Prognostic Rating Scale to the Rorschach.

A decrease in pathology was suggested by the three measures which were employed. The beneficial effects of reserpine were more pronounced for the group which had received reserpine initially than for the group which had received reserpine after a period of placebo treatment. It was further suggested by these measures that patients changed from reserpine to placebo do not maintain the gains made on the drug.

The relatively greater gains for the reserpine group during the first 7-week period suggest the importance of non-drug factors which must be taken into account in any subsequent research.

(Author's Abstr.)

MAY

The Role of Genetics in Psychiatry. *Rainer, J. D., and Kallmann, F. J.* 403
*Skin Histamine Reaction in Schizophrenic and Non-Schizophrenic Mental Patients. *Weckowicz, T. E., and Hall, R.* 415

Hallucinations of Poliomyelitis Patients During Treatment in a Respirator. *Mendelson, J., et al.* 421
The Impact of Psychoanalysis Upon Child Psychology. *Escalona, S.* 429
A Comparison of Psychoanalysis With the Dynamic Psychotherapies. *Murphy, W. F.* 441
*The Phenomenon of Auditory Hallucinations in Chronic Alcoholism. *Victor, M., and Hope, J. M.* 451

Skin Histamine Reaction in Schizophrenic and Non-Schizophrenic Mental Patients

Schizophrenics can be differentiated by skin histamine reaction from other patients only as a group. There are some individual schizophrenics who have a strong histamine skin reaction and some individual non-schizophrenic patients who have a weak histamine skin reaction. The hope that it would be possible to develop a specific skin histamine test for schizophrenia appears to have been too sanguine. All tests, either physiological or psychological, for schizophrenia so far attempted, managed to differentiate schizophrenics as a group, but failed to diagnose the individual patients. One can refer to these tests as being peripheral, because they measure, usually indirectly, only secondary (dependent) variables. The real diagnostic test for schizophrenia is yet to come. It will probably coincide with the discovery of the key metabolic disorder responsible for schizophrenic illness.

(Authors' Abstr.)

The Phenomenon of Auditory Hallucinations in Chronic Alcoholism

1. The phenomenon of auditory hallucinations in chronic alcoholism has been studied. Clinical observations of 76 instances of this illness are recorded and illustrative case histories are presented.

2. Although the occurrence of auditory hallucinations was the outstanding feature in all the cases, several clinical forms could be distinguished: (*a*) in 68 instances the hallucinations were benign and transient and were frequently associated with other signs of prolonged inebriation and abstinence; (*b*) in 8 cases the hallucinations persisted for several weeks or months; (*c*) in 4 of these the illness was indistinguishable from the first type at its outset, but later the characteristic symptoms of schizophrenia developed.

3. Our observations indicate that a syndrome may emerge in relationship to excessive drinking which is clinically indistinguishable from schizophrenia. The patients with this syndrome, prior to its appearance, could not be considered schizophrenics and the age of onset was later than that usually encountered in schizophrenia. The concept, that the schizophrenic-like illness is simply "latent schizophrenia made manifest by alcohol" is neither logical nor consistent with the observed facts.

4. The current theories concerning the aetiology of auditory hallucinosis are critically evaluated. Data are presented to show that the development of auditory hallucinations depends not only on the effect of prolonged inebriation, but ultimately on the effect of withdrawal of alcohol. The factor or factors responsible for the prolongation of the illness and for the occasional transition to a schizophrenic-like form, are unknown.

(Authors' Abstr.)

JUNE

Psychological and Phenomenological Aspects of Disorders of the Body Image. *Gerstmann, J.* 499
On the Nature of Uncivilized and Civilized People. *Doob, L. W.* 513
A Multi-Generation Approach to Treatment of Psychopathology. *Mendell, D., and Fisher, S.* 523
*Hexafluorodiethyl Ether (Indoklon): Its Use as a Convulsant in Psychiatric Treatment. *Esquibel, A. J., et al.* 530
Prevention of Anoxia During Succinylcholine-Electroshock Therapy by the Use of a New Treatment Technique. *Michael, K. D., and Wunderman, D. C.* 535
A Comparison of Two Kinds of Flicker Phenomena as Indicators of CNS Involvement. *McGuire, F. L.* 540
Dynamic Aspects of Psychiatric Symptoms Among Prisoners in Terms of Aggression. *Rinsley, D. B.* 547
Examining the Offender: Some Procedures and Peculiarities. *Murphy, B. W.* 554
Relationships in the Manifest Content of Dreams Occurring on the Same Night. *Dement, W., and Wolpert, E. A.* 568
The Seeking of Sympathy or Pity. *Hollender, M. H.* 579

Hexafluorodiethyl Ether (Indoklon): Its Use as a Convulsant in Psychiatric Treatment

Hexafluorodiethyl ether was administered to 60 of 120 patients randomly selected for convulsive therapy. In the 720 treatments administered to these 60 patients there was no failure in bringing about a *grand mal* convulsion. Also, there was no evidence of any change in sensitivity or tolerance to the standard convulsive dose with progressive treatment. The convulsive seizure was similar to that produced by electric stimulation, except for a more gradual onset. There appeared to be less immediate post-convulsive anoxia, and immediate post-convulsive psychomotor activity appeared diminished in comparison with E.C.T. Subjectively, a majority of those patients who had been exposed to both types of treatment felt less apprehensive with the inhalation procedure. From these results it appears that Indoklon is an effective and safe convulsive agent that can be administered safely and conveniently on a large scale basis.

(Authors' Abstr.)

VOL. 127 JULY, 1958

*The Limbic System with Respect to Self-Preservation and the Preservation of the Species. *MacLean, P. D.* 1
*Circulating Epinephrine and Norepinephrine in Changing Emotional States. *Regan, P. F., III, and Reilly, J.* 12
A Comparative Study of Chlorpromazine and Triflupromazine in the Management of the Chronic Hospitalized Psychotic Patient. *Hanlon, T. E., et al.* 17
The Pathophysiology of Grand Mal Seizures Generalized from the Start. *Gastaut, H.* 21
Hypereridism and Attempted Suicide in Chinese. *Yap, P. M.* 34
New Symptoms of Closing-In and Their Interpretation. *Vereecken, P. J.* 42
Some Psychiatric Observations of Ulcerative Colitis with Report of a Case. *Joseph, E. D., et al.* 51
Schizophrenia and Related Problems. *Branch, C. H. H.* 58
What Do Schizophrenics Learn During Psychotherapy and How Do They Learn It? *Brody, E. B.* 66
Patterns and Course of Schizophrenia in the Deaf. *Altshuler, K. Z., and Rainer, J. D.* 77
A Consideration of the Therapeutic Use of Music in Psychiatric Illness. *Sherwin, A. C.* 84

The Limbic System with Respect to Self-Preservation and the Preservation of the Species

The greater part of the phylogenetically old cortex is contained in the great limbic lobe of Broca. This lobe is found as a common denominator in the brains of all mammals. Together with its subcortical cell stations it comprises a functionally integrated system, which, in keeping with Broca's terminology, may be referred to as the *limbic system.* Evidence is cited which indicates that the frontotemporal portion of the limbic system is largely concerned with mechanisms that are involved in obtaining and assimilating food and are thereby essential to *self-preservation.* Attention is then turned to recent findings that are beginning to suggest that another portion of the limbic system, involving parts of the septum, hippocampus and cingulate gyrus, is concerned with expressive and feeling states that are conducive to the *preservation of the species.* Some of the implications of these new findings in regard to a number of provocative neurological questions are discussed.

(Author's Abstr.)

Circulating Epinephrine and Norepinephrine in Changing Emotional States

In a study of 60 hospitalized psychiatric patients, it was found that circulating levels of epinephrine and norepinephrine tend to change in accordance with changes in an individual's emotional state. The presence or absence of certain descriptive and personality features indicates the frequency with which this parallel relationship will occur. Through an analysis of these factors, it seems possible to distinguish subgroups of the population in whom changes in epinephrine or norepinephrine, or both, tend to be reliable indicators of similar changes in the patient's emotional status.

(Authors' Abstr.)

AUGUST

Experimental Interference With Reality Contact (Perceptual Isolation): Method and Group Results. *Goldberger, L., and Holt, R. R.* 99
The Affect System: Foresight and Fantasy. *Rosenzweig, N.* 113
Recall of Some Phenomena of Sleep. *Tapia, F., et al.* 119
Psychodynamic Relationships Between the Degree of Anxiety and Other Clinical Symptoms. *Lesse, S.* 124
Changes in the Symbolic Process During a Psychoanalytic Treatment. *Seidenberg, R.* 131
Personality Determinants of Medical Clinic Consultation. *Roessler, R., and Greenfield, N. S.* 142
Day Hospital Care and Changing Family Attitudes Toward the Mentally Ill. *Lystad, M. H.* 145
The Speech Patterns of Schizophrenic Patients: A Method of Assessing Relative Degree of Personal Disorganization and Social Alienation. *Gottschalk, L. A., et al.* 153
Influence of Chlorpromazine on Certain Biochemical Variables of Chronic Male Schizophrenics. *Mefferd, R. B., Jr., et al.* 167
Suggested Biochemical Factors in Schizophrenia. *Ovshinsky, S. R.* 180
*Preliminary Study of a New Anti-Depressant Drug. *Friedhoff, A. J., et al.* 185
Effects of Electroshock Therapy on the Excretion of Epinephrine. *Sourkes, T. L., et al.* 191

Preliminary Study of a New Anti-Depressant Drug

1. In this controlled study thirty-two schizophrenic patients were given various doses of SKF #5 without significant improvement.

2. Twenty-five seriously depressed patients were treated with this drug and improvement was noted in 76 per cent. of the group.

3. The development of high levels of alkaline phosphates in twelve patients suggests that further appraisal of these findings should be done in a longer range study.

4. The observable side effects were not of a serious nature.

5. The effective dose appears to be between 60 and 300 mg. per day. Initial doses should be as low as 25 to 30 mg. per day.

(Authors' Abstr.)

VOL. 21 J. NEUR. NEUROSURG. PSYCHIAT. FEBRUARY, 1958

The Relative Merits of Encephalography and Ventriculography for the Investigation of Intracranial Tumours. *Norlén, G., and Wickbom, I.* 1

Ischaemic and Post-ischaemic Numbness and Paraesthesiae. *Nathan, P. W.* 12

The Surgical Prognostic Significance of the Electroencephalographic Prediction of Ammon's Horn Sclerosis in Epileptics. *Kennedy, W. A., and Hill, D.* 24

A Report of Two Cases of the Juvenile Form of Amaurotic Familial Idiocy (Cerebro-macular Degeneration). *Jefferson, M., and Rutter, M. L.* 31

Spinal Angioma with Symptoms in Pregnancy. *Newman, M. J. D.* 38

Pulsating Exophthalmos in von Recklinghausen's Disease. *Langmaid, C., and Daws, A.* 42

Optic Atrophy and Nerve Deafness in Diabetes Mellitus. *Shaw, D. A., and Duncan, L. J. P.* 47

Sodium Amytal and Extraversion. *Laverty, S. G.* 50

A Controlled Trial of Methyl Phenidate (Ritalin) in the Treatment of Depressive States. *Robin, A. A., and Wiseberg, S.* 55

Epinephrine Metabolites and Pigmentation in the Central Nervous System in a Case of Phenylpyruvic Oligophrenia. *Fellman, J. H.* 58

MAY

The Neuropathy of Multiple Myeloma. *Victor, M., et al.* 73

The Relapsing Course of Certain Meningiomas in Relation to Pregnancy and Menstruation. *Bickerstaff, E. R., et al.* 89

A Case of Intramedullary Neurinoma. *Ramamurthi, B., et al.* 92

Recurrent Multiple Cranial Nerve Palsies. *Symonds, C.* 95

Finger Tremor in Tabetic Patients and Its Bearing on the Mechanism Producing the Rhythm of Physiological Tremor. *Halliday, A. M., and Redfearn, J. W. T.* .. 101

Sensory Nerve Action Potentials in Patients with Peripheral Nerve Lesions. *Gilliatt, R. W., and Sears, T. A.* 109

The Psycho-galvanic Reflex: A Comparison of A.C. Skin Resistance and Skin Potential Changes. *Montagu, J. D.* 119

Clinical Aspects of Susceptibility to Methylpentynol. *Marley, E., and Bartholomew, A. A.* 129

An Investigation of the Rosenow Antibody Antigen Skin Reaction in Schizophrenia. *Gurassa, W. P., and Fleischhacker, H. H.* 141

Multilocular Cystic Encephalopathy of Infants. *Crome, L.* 146

VOL. 17 J. NEUROPATH. EX. NEUR. APRIL, 1958

An Autoradiographic Demonstration of the Relative Vascularity of the Central Nervous System of the Cat with Iodine 131-Labeled Serum Albumin. *Barlow, C. F., et al.* 191

5-Nucleotidase in Normal and Diseased Human Skeletal Muscle. *Beckett, E. B., and Bourne, G. H.* 199

Progressive Deteriorating Diseases of Infancy. First Communication. *Benda, C., and Melchior, J. C.* 205

Study on the Reticulin Fibers of Brain Tumors. *Bölönyi, F.* 240

The Tuberous Sclerosis Complex. *Cares, R. M.* 247

Some Observations on the Congenital Deformity of the Central Nervous System Known as the Arnold-Chiari Malformation. *Daniel, P. M., and Strich, S. J.* 255

Biochemistry in Routine Neuropathology. I. Quantitative Chemical Determinations in Formaldehyde Preserved Brains as an Aid in the Neuropathology of Demyelinating Conditions and Lipidoses. *Edgar, G. W. F.* 267

Biochemistry in Routine Neuropathology. II. Determination of Hexosamine in Chloroform-Methanol Extracts of Brain as a Diagnostic Procedure with Special References to the Quantitation of Gangliosides. *Smits, G., and Edgar, G. W. F.* 269

*Studies on Multiple Sclerosis. I. Multiple Sclerosis Viewed as a "Chronic Disseminated Encephalomyelitis". II. Etio-Pathogenesis of Multiple Sclerosis (Infectious Allergic or Toxic Allergic). *Ferraro, A.* 278

Lesions in the Anterior Motor Horn Cells of Rats after Administration of Bis-β-Cyanoethylamine. (Study of a Nitrile Closely Related to the Lathrus Factor). *Hartmann, H. A., et al.* 298

Hypophyseal Duct Tumors (Craniopharyngiomas) Versus Adamantinomas. *Hirsch, O.* 305

Patho-Anatomic and Histopathologic Character of Lesions in the Central Nervous System in Man Following Anti-Rabic Treatment. *Jones, E.* 315

Cortical Blindness. *Madow, L.* 324
Toluidine Blue Staining Reactions in Superior Cervical and Nodose Ganglia Following Stimulation and Drug Administration. *Richins, C. A., and Hall, J. L.* 333
A Clinico-Anatomic Study of Personality and Mood Disturbances Associated with Gliomas of the Cerebrum. *Riggs, J. E., and Rupp, C.* 338
*The Histologic Structure of the "Inclusion Bodies" of the Neurons in Pick's Disease. A Study with Silver Carbonate. *Scharenberg, K.* 346
Polioencephalomyelopathy Reminiscent of Creutzfeldt-Jakob's Syndrome. A Clinico-Pathologic Report of a Case. *Schwarz, G. A., and Barrows, L. J.* 352
The Meningiomas of the Lateral Ventricles of the Brain. *Tukanowicz, S. A., and Grant, F. C.* 367
Degeneration of the Brain Stem Reticular Formation. Other Parts of the Brain Stem and the Cerebellum. An Example of Heterogenous Systemic Degeneration of the Central Nervous System. *Verhaart, W. J. C.* 382
A Study of Fifty Intracranial Vascular Tumors Found Incidentally at Necropsy. *White, R. J., et al.* 392
The *In Vitro* Action of Some Organic Acids on Myelin. *Wolfgram, F., and Rose, A. S.* 399

Studies on Multiple Sclerosis. I. Multiple Sclerosis Viewed as a "Chronic Disseminated Encephalomyelitis". II. Etio-Pathogenesis of Multiple Sclerosis (Infectious Allergic or Toxic Allergic)

No appreciable clinical differences seem to exist in support of a differential diagnosis between acute multiple sclerosis, the early stages of multiple sclerosis, and the acute or sub-acute varieties of post-exanthematic, post-vaccinal, or post-infectious encephalomyelitides.

The value of relapses and remissions which in the past was considered an important differential feature favouring multiple sclerosis, has lost its importance in the face of relapses and remissions occurring also in various types of acute disseminated encephalomyelitides.

From a pathological standpoint no appreciable differences exist between the lesions of acute disseminated encephalomyelitides, the post-vaccinal ones, acute multiple sclerosis and the acute foci related to relapses in chronic multiple sclerosis.

It is therefore proposed to label multiple sclerosis as a "chronic variety of disseminated encephalomyelitis". Multiple sclerosis may then represent the chronic stage of any acute disseminated encephalomyelitis and not a transformation of a disease into another one.

The general biological factors which determine in some cases a simple acute and reversible encephalomyelitic process and in others a chronic progressive process leading to irreversible glia scars are the expression of the immuno-biological constitution which governs reactions of individuals or groups of individuals, to various noxi. The organization of individual immuno-biological defenses is the result of constitutional hereditary factors, modified by age, environment and adaptation.

Acute disseminated encephalomyelitides and multiple sclerosis have a common aetio-pathogenesis. This the author would like to label "Infectious-allergic" or "Toxic-allergic".

The toxic etiologic component of the combination, as well as of the infectious one, may be different in various groups of cases, just as it is different in measles, scarlet fever, variola, varicella or influenza. The toxic or infectious etiologic components must combine with a partial antigen in order to create a complete "demyelinating encephalitogenic antigen". This complete antigen will trigger off an allergic reaction. The ultimate pathogenic mechanism responsible for the clinico-pathologic changes will thus remain an allergic one.

This concept of "infectious-allergic", or "toxic-allergic" etio-pathogenesis of "acute and chronic disseminated encephalomyelitis" is an elaboration of the author's earlier general concept (1944) that the pathology of multiple sclerosis could be viewed in the light of an "allergic reaction of the central nervous system".

We do not know under what biochemical or immuno-biological circumstances the combination of an infectious or toxic agent and partial antigens occurs. Factors of our individual immuno-biological organization and related protective mechanism must play an important role toward that combination, as well as toward the type of pathological reaction leading to an acute transitory or to a chronic type of disseminated encephalomyelitis. Very little is also known about possible mutations of viruses or bacilli which, particularly in the case of atypical tuberculosis, may play an important role in the type of pathologic reaction of the nervous tissue related to "toxic-allergic" or "infectious-allergic" etio-pathogenesis of multiple sclerosis.

(Author's Abstr.)

The Histologic Structure of the "Inclusion Bodies" of the Neurons in Pick's Disease. A Study with Silver Carbonate

The complex histopathologic picture of degeneration of neurons is broken down into its component parts by the silver carbonate technic, so that various degenerative types can be recognized. The outstanding histopathologic phenomenon is the transformation of the intra-cellular fibers into amorphous material called "inclusion bodies" which fully confirms the conclusion of Alzheimer (2). However, the degeneration of the intracellular structures is not confined to the fibers, does not run a uniform course and does not always culminate in the formation of amorphous inclusions. The latter phenomenon is least conspicuous in Type I in which the entire system of fibers is transformed into a coarse network.

Type II on the contrary, is characterized by one or more sharply outlined "inclusion" bodies in the cytoplasm, some of which are surrounded by a ring of fragments or a halo.

In Type III the neurons are greatly enlarged, contain numerous vacuoles filled with a substance which stains reddish brown with oil red "O"; it probably is a lipoid. In more advanced stages the cells become homogeneously stained brown red or blue black, the vacuoles disappear and no histologic details can be recognized.

Type IV represents the final stage of destruction of nerve cells which are reduced to amorphous fragments and have lost all semblance of normal structure.

Disintegration of the nuclei of the neurons is less conspicuous than that of the cytoplasm because the former are frequently destroyed early in the course of degeneration; those which survive are swollen and blurred, and are partly extruded from the cell. In spite of advanced degeneration, numerous dendrites are preserved.

The degeneration of the neurons overshadows other histopathologic changes to such an extent that it cannot be doubted that Pick's disease is a primary disease of the parenchyma. The effects of destruction of neurons on the myelo- and glia-architecture are profound, but cannot be discussed here beyond stating that there are also "inclusion bodies" in the astroglia and that various degenerative changes occur in the oligodendroglia. There are usually numerous microglia elements in the form of gitter cells, many of them are laden with fat.

(Authors' Abstr.)

JULY

A Systematic Analysis of Intracranial Aneurysms from the Autopsy File of the Presbyterian Hospital, 1914 to 1956. *Housepian, E. M., and Pool, J. L.* 409
Aneurysm of the Vein of Galen. *Hirano, A., and Terry, R. D.* 424
Studies of White Matter. I. Chemical Constitution and Respiration of Neuroglial and Myelin Enriched Fractions of White Matter. *Korey, S. R., et al.* 430
Studies in White Matter. II. Encephalitogenic Activity of Myelin Rich Fraction and Neuroglial Concentrate of White Matter. *Scheinberg, L. C., and Korey, S. R.* .. 439
*Diffuse Traumatic Degeneration of the Cerebral Gray Matter. *Denst, J., et al.* .. 450
Hemiplegia in Early Life Associated with Thrombosis of the Sagittal Sinus and its Tributary Veins in One Hemisphere. *Dekaban, A., and Norman, R. M.* 461
Giant-Cell Arteritis Involving Small Meningeal and Intracerebral Vessels. *McCormick, H. M., and Neubuerger, K. T.* 471
Simultaneous Uptake of Radioactive Copper and Radioactive Potassium by Rat Brains. *Spencer, R. P., and Mitchell, T. G.* 479
Cerebral Arachnoid Cysts. *Starkman, S. P., et al.* 484
Gliomatous Polio- and Leucodystrophy in a Young Child. *de Vries, E.* 501
A Simple Method for Preparation of Gross Brain Sections. *Settlage, P. H., et al.* .. 510
American Association of Neuropathologists, Annual Meeting, Atlantic City, N.J. .. 514

Diffuse Traumatic Degeneration of the Cerebral Gray Matter

A diffuse degeneration of the cerebral gray matter is described in a 14-year-old boy who died 10 months after closed traumatic injury to the skull. Extensive cortical devastation and gliosis with selective involvement of the central gray matter and without evidence of crude traumatic destruction of hemorrhage were the outstanding features. The parenchymal lesions appeared to be related closely to the concussive force; a constellation of factors which included edema, thixotropy, and vasal disturbances were undoubtedly important in the pathogenesis.

(Authors' Abstr.)

VOL. 21 J. NEUROPHYSIOL. JULY, 1958

Crossed Inhibition of Sacral Motoneurones. *Curtis, D. R., et al.* 319
Strychninization of Bulbar Ventromedial Reticular Formation and Galvanic Skin Reflex in Thalamic Cats. *Wang, G. H.* 327
Synaptic Transmission in Spinal Cord During Wallerian Degeneration of Dorsal Root Fibers. *Vera, C. L., and Luco, J. V.* 334
Evoked Central Nervous System Activity Relating to Peripheral Unmyelinated or "C" Fibers in Cat. *Collins, W. F., and Randt, C. T.* 345
Effects of Discrete Brainstem Lesions in Cats on Perception of Noxious Stimulation. *Melzack, R., et al.* 353
Spontaneous Activity of Betz Cells in Cats with Midbrain Lesions. *Martin, A. R., and Branch, C. L.* 368
Inhibition of Betz Cell Activity by Thalamic and Cortical Stimulation. *Branch, C. L., and Martin, A. R.* 380
Effect of Striatal and Claustral Stimulation Upon Spinal Reflex and Strychnine Activity. *Segundo, J. P., et al.* 391
Subthalamic Hyperkinesia in Rhesus Monkey. Effects of Secondary Lesions in Red Nucleus and Brachium Conjunctivum. *Carpenter, M. B., and Brittin, G. M.* .. 400

SEPTEMBER

Mechanism of Lateral Inhibition in Eye of *Limulus*. *Tomita, T.* 419
Electroclinical Features of Convulsions Induced by Stimulation of Brain Stem. *Kreindler, A., et al.* 430
Potentials Evoked in Cat Cerebral Cortex by Diffuse and by Punctiform Photic Stimuli. *Doty, R. W.* 437
Modulation of Sensory Discharges by Efferent Spindle Excitation. *Eyzaguirre, C.* .. 465
Representation of Cutaneous Tactile Sensibility in Cerebral Cortex of *Cebus*. *Hirsch, J. F., and Coxe, W. S.* 481
Thalamocortical Relations as Revealed by Induced Slow Potential Changes. *Brookhart, J. M., et al.* 499

VOL. 15 J. NEUROSURG. JULY, 1958

Dislocation of the Atlas on the Axis. The Value of Early Fusion of C1, C2, and C3. *Alexander, E., Jr., et al.* 353
Carotid Artery Compression as Employed Both in the Past and in the Present. *Webster, J. E., and Gurdjian, E. S.* 372
Saccular Aneurysms of the Posterior Fossa. *De-Saussure, R. L., et al.* 385
Cavernous Hemangiomas of the Cerebral Hemispheres. *Schneider, R. C., and Liss, L.* 392
Arterial Aneurysms of the Internal Carotid Artery and its Bifurcation. An Analysis of 69 Aneurysms Treated Mainly by Direct Surgical Attack. *af Björkesten, G.* .. 400
Positron-Scanning with Copper-64 in the Diagnosis of Intracranial Lesions. Partition of Copper-64 Versenate in, and Excretion from, the Body. *Bagnall, H. J., et al.* 411
Surgical Reconstruction of Occluded Cervical Carotid Artery. Report of a Successful Case with 4-Year Follow Up and Three Examples without Such Treatment. *Hamlin, H., et al.* 427
Complications in the Surgery of Internal Carotid Artery Thrombosis. *Lemmen, L. J., et al.* 438
Experience with Surgical Treatment in Twenty Cases of Pituitary Adenomas. *Echols, D. H.* 447

SEPTEMBER

Signs and Symptoms of Supratentorial Arteriovenous Aneurysms. *Tönnis, W., et al.* 471
Pituicytoma. Tumor of the Sella Turcica. A Clinicopathological Study. *Liss, L., and Kahn, E. A.* 481
Glioblastoma Multiforme. Review of 219 Cases with Regard to Natural History, Pathology, Diagnostic Methods, and Treatment. *Frankel, S. A., and German, W. J.* 489
Hypophysectomy in Severe Diabetes. I. Neurosurgical Aspects. *Javid, M., et al.* .. 504
Pyogenic Neurosurgical Infections in Korean Battle Casualties. *Wannamaker, G. T., and Pulaski, E. J.* 512
Destruction of the Hypophysis with Radioactive Colloidal Chromic Phosphate in Cancer of the Prostate. *Johnson, P. C., et al.* 519
A Study of the Treatment of 637 Patients with Trigeminal Neuralgia. *Ruge, D., et al.* 528
Postoperative Bone Changes Following Lumbar Disc Removal. *Schultz, E. C.* .. 537
A New Theory on the Dynamics of Brain Concussion and Brain Injury. *Gross, A. G.* 548

VOL. 26 J. PERSONAL. JUNE, 1958

Organizational Characteristics of Social Judgment: A Developmental Investigation. *Gollin, E. S.* 139
Some Motivational Factors in Cooperation and Competition. *Shaw, M. E.* 155
Some Psychological Correlates of Worldmindedness and Authoritarianism. *Smith, H. P., and Rosen, E. W.* 170
Electrophysiological Correlates of Intelligence. *Mundy-Castle, A. C.* 184
Surveillance and Trust. *Strickland, L. H.* 200
Some Personality Correlates of Learning Without Verbal Awareness. *Eriksen, C. W., et al.* 216
Conformity and the A-B-X Model. *Steiner, I. D., and Peters, S. C.* 229
Dispositional Effects Upon Conformity at Different Levels of Discrepancy. *Schroder, H. M., and Hunt, D. E.* 243
Differential Movie-Viewing Behavior of Male and Female Viewers. *Maccoby, E. E., et al.* 259
The Cognitive Significance of Rorschach Developmental Scores. *Brooks, M. O., and Phillips, L.* 268
Rorschach Scores and Decontextualization: A Developmental View. *Lipton, H., et al.* 291

SEPTEMBER

The Influence of Anxiety Upon Learning: Interference or Drive Increment? *Nicholson, W. M.* 303
The Relationship of Body Image Distortions to Body Reactivity Gradients. *Fisher, S., and Abercrombie, J.* 320

Emational Tension and the Generality of Its Effect Upon Intellectual Performance. *Bevan, W., and Maier, R. A.* 330
Perception of Apparent Movement in Korsakoff's Psychosis. *Talland, G. A.* 337
Cognitive Style and Stimulus Ambiguity. *Loomis, H. K., and Moskowitz, S.* 349
Individual and Sex Differences in Cortical Conductivity. *Barthol, R. P.* 365
The Effects of Sets Induced by Labeling on the Modification of Attitudes. *Di Vesta, F. J., and Bossart, P.* 379
Autonomic Feedback: A Correlational Study. *Mandler, G., and Kremen, I.* 388
Group Support, Legitimate Power, and Social Influence. *Raven, B. H., and French, J. R. P., Jr.* 400
An Investigation of the Psychoanalytic Theory of Paranoid Delusions. *Zamansky, H. S.* 410
Time Imagery and the Achievement Motive. *Knapp, R. H., and Garbutt, J. T.* .. 426

VOL. 22 J. PROJECT. TECHNIQ. MARCH, 1958

Some Relationships Among Thematic Projective Tests of Various Degrees of Structuredness and Behavior in a Group Situation. *Edgar, C. L., and Shneidman, E. S.* 3
The Development and Applications of the Children's Insight Test. *Engel, M.* .. 13
Psychological Testing with the Older Client: A Case of Paresis. *Gruen, A.* 26
Male Homosexuality in the Rorschach. *Hooker, E.* 33
The Differential Effects of Negated Word Associations on Ability to Recall "Traumatic" and "Non-Traumatic" Stimulus Words. *Jones, R. M.* 55
Stress, Anxiety and Response Complexity on the Rorschach Test. *Kates, S. L., and Schwartz, F.* 64
Rating Scales for Ego Functioning Applicable to Diagnostic Testing. *Klopfer, B., et al.* 70
Three Blind Interpretations of a TAT Record. *Lyle, J., et al.* 82
Personal Significance of Rorschach Figures. *Richards, T. W.* 97
On the Psychoanalytic Study of Retest Results. *Schafer, R.* 102

JUNE

Henry A. Murray—An Appreciation. *Bellak, L.* 143
Use of the TAT in Assessment of the Personality Syndrome of Alienation. *Davids, A., and Rosenblatt, D.* 145
Affective Complexity and Psychotherapy: Some Comparisons of Time-Limited and Unlimited Treatment. *Henry, W. E., and Shlien, J. M.* 153
Formal Aspects of the TAT—A Neglected Resource. *Holt, R. R.* 163
Thematic Apperception Test: The Strategy of Research. *Lindzey, G.* 173
How Was This Story Told? *Schafer, R.* 181
TAT Heroes of Suicidal and Non-Suicidal Subjects. *Shneidman, E. S., and Farberow, N. L.* 211
A Principle for the Interpretation of Fantasy. *Wyatt, F.* 229

SEPTEMBER

Suggestions for Research with Projective Techniques. *Blank, L.* 263
Development and Standardization of the Group Personality Projective Test. *Cassel, R. N., and Kahn, T. C.* 267
The Rorschach Records of Suicidal Patients. An Application of a Comparative Matching Technique. *Costello, C. G.* 272
The Relationship Between EEG Abnormality, Some Psychological Factors and Delinquent Behavior. *Foster, A. L.* 276
A Critique of Shneidman and Farberow's "TAT Heroes of Suicidal and Non-Suicidal Subjects". *Friedman, I.* 281
Projective Tests as Measures of Defensive Activity. *Heath, D.* 284
Stimulus-Values of Rorschach Cards for Children. *Levy, E.* 293
A Factor Analysis of the WISC and Rorschach. *Lotsof, E. J., et al.* 297
Early Recollections as a Projective Technique. *Mosak, H. H.* 302
The Eavesdropping Questions: A New Projective Technique to Aid in Determining Self-Image. *Newstrand, M. B.* 312
A Comparison of Copied and Recalled Reproductions of the Bender-Gestalt Designs. *Olin, T. D., and Reznikoff, M.* 320
Some Psychosexual Differences Between Kibbutz and Non-Kibbutz Israeli Boys. *Rabin, A. I.* 328
The Effect of Type of Administration and Sex of Subject on Emotional Tone and Outcome Ratings of TAT Stories. *Sarason, B. R., and I. C.* 333
The Influence of Artistic Quality on Judgments of Patient and Non-Patient Status from Human Figure Drawings. *Sherman, L. J.* 338
Clinical Correlates of Facilitation and Inhibition of Response to Emotional Stimuli. *Ullmann, L. P.* 341

VOL. 45 J. PSYCHOL. JANUARY, 1958

Drugs and Human Fatigue: *GSR* Parameters. *Burch, N. R., and Greiner, T. H.* 3
The "Dynamic-Affective" Phase in the Development of Concepts. *Honkavaara, S.* 11
A Critical Re-evaluation of the Color and Form Reaction, and Disproving of the Hypotheses Connected with It. *Honkavaara, S.* 25
An Attempt to Measure Intelligence in Terms of Organic Development. *Honkavaara, S.* 37
Some Critical Notes Concerning the Concept of Rigidity and Its Measurement. *Honkavaara, S.* 43
Nutritional Deficiencies and Emotional Disturbances. *Bell, E. C.* 47
The Stablemate Concept of Therapy as Affected by *LSD* in Schizophrenia. *Abramson, H. A., et al.* 75
The Movement of Ground Over Figure: A New Form of Apparent Movement. *Barthol, R. P.* 85
The Place of Private Practice in Professional Psychology. *Meltzer, H.* 93
The Humascope: A Modified Stereoscope. *Adlerstein, A. M.* 109
The Concept of "Hypnosis". *Barber, T. Z.* 115
The Construction of a Test Battery for Counseling Mature Individuals with Psychological Retirement Problems. *Freeman, G. L., and Taylor, E. K.* 133

APRIL

Lysergic Acid Diethylamide (LSD-25): XXV. Effect on Social Order of Newts, *Triturus v. viridescens (Raf.). Evans, L. T., and Abramson, H. A.* 153
A Guide to Published Literature Relating to the Authoritarian Personality Through 1956. *Christie, R., and Cook, P.* 171
The Magnetic Board Rating Technique. *Rettig, S., et al.* 201
Body Image Boundaries and Sexual Behavior. *Fisher, S., and Cleveland, S. E.* 207
Figure-Ground Phenomena in the Perception of Physical and Social Stimuli. *Rudin, S. A., and Stagner, R.* 213
Anxiety, Stress, and Thinking: An Experimental Investigation. *Dowis, J. L., and Diethelm, O.* 227
The Dynamics of Hypnotically Induced Anti-Social Behavior. *Kline, M. V.* 239
Color Coded Multiple-Image Photography for Studying Related Rates of Movement. *Jones, F. P., et al.* 247
Perceptual Change Occurring During Psychotherapy: Three Clinical Histories. *Rudin, S. A.* 253
Form Perception and Size Constancy in the Duckling. *Pastore, N.* 259
Lysergic Acid Diethylamide (LSD-25): XXVI. Effect on Social Order of the Fighting Fish, *Betta splendens. Evans, L. T., et al.* 263
Changes in the Level of Blood Sugar and Sensory and Motor Performance Brought About by Insulin Coma Therapy. *Landis, C., et al.* 275
Two Generalized Ratio Scaling Methods. *Ekman, G.* 287

VOL. 46 JULY, 1958

Organization Process in Perception as a Measure of Intelligence. *Honkavaara, S.* 3
The Accuracy of Perception in Relation to the Color and Form Reaction. *Honkavaara, S.* 13
The Accuracy of Perception in Relation to Interpersonal Relationships. *Honkavaara, S.* 19
Comparison of the Relation of Color- and Form-Reactors at Harvard and London Universities. *Honkavaara, S.* 23
Relation of Interpersonal Preference and Emotional Attitude of the Subjects. *Honkavaara, S.* 25
The Color and Form Reaction as a Basis of Interpersonal Relationships. *Honkavaara, S.* 33
The Color and Form Reaction as a Basis for Differential Psychotherapeutic Approach. *Honkavaara, S.* 39
Binocular Fusion of Dissimilar Figures. *Engel, E.* 53
Relation Between the Height-Width Ratio and the Perception of Types of Movement in Lissajou Figures. *Philip, B. R.* 59
A Comparative Study of the Onset of Menarche Among Negro and White Children. *Henton, C. L.* 65
An Analytical Review of the Experimental Basis of Subception. *Naylor, J. C., and Lawshe, C. H.* 75
On the Psychological Study of Small Whales. *Kellogg, W. N.* 97
Studies in Physiognomic Perception: V. Effect of Ascending and Descending Gliding Tones on Autokinetic Motion. *Miller, A., et al.* 101
Some Factors Influencing Critical Flicker Frequency. *Bartley, S. H.* 107
The Perception of the Blind Spot and the Convergence of the Visual Pathways. *Wilkinson, F. R., and Fleming, D. E.* 117
A High-Level Interest-Values Reference Test for Counselling Pre-Retirants. *Freeman, G. L.* 121
Folie à Deux in Husband and Wife. *Dawson, J. G., and Burke, G. W.* 141

Participation in Leisure Time Activities as Related to Selected Vocational and Social Variables. *Standlee, L. S., and Popham, W. J.* 149
Theory and Evidence of Imprinting in Human Infants. *Gray, P. H.* 155
A Factorial Analysis of Behavior Ratings of Hospitalized Mental Patients. *Bryant, J. H., et al.* 167
Clinical Implications of Factors Derived from the Southeast Louisiana Hospital Rating Scale. *Dawson, J. G., et al.* 175

VOL. 47 **J. SOC. PSYCHOL.** FEBRUARY, 1958

Arab Students' Choices of Ways to Live. *Prothro, E. T.* 3
The Intellectual Functioning of Delinquents. *Blank, L.* 9
The Effect of Relevant and Non-Relevant Sources on Attitude Change. *Kerrick, J. S.* 15
Toward a New Instinct Theory. *Oppenheimer, O.* 21
Some Effects of Irrelevant Information Upon Problem-Solving by Small Groups. *Shaw, M. E.* 33
A Method of Analysis of the Process of Verbal Communication Between a Group of Emotionally Disturbed Adolescents, and Their Friends and Relatives. *Salzinger, K.* 39
Intellectual Inhibition and the Search for Scientific Truth. *Blanchard, W. H.* 55
The Utility of the Shevky Typology for the Design of Urban Sub-Area Field Studies. *Bell, W.* 71
Experimental Results in Relation to a Mathematical Theory of Behavioral Diffusion. *Winthrop, H.* 85
Individual Differences in Evaluations of Group Discussions as a Projective Measure of Attitudes Toward Leadership. *Campbell, D. T., and Mehra, K.* 101
Semantic Idiosyncracy as a Method in the Study of Attitudes. *Campbell, D. T., and Shanan, J.* 107
The Sense of Effectiveness and Response to Public Issues. *Douvan, E.* 111
Student Ratings of Popularity and Scholastic Ability of Their Peers and Actual Scholastic Performances of Those Peers. *Johnson, E. E.* 127
The Effect of Religious Background on Modes of Handling Anger. *Bateman, M. M., and Jensen, J. S.* 133

MAY

Preface II to the Continued Immediate Publication of Cross-Cultural Research. *Murchison, C.* 157
Objectives and Methods of African Psychological Research. *Biesheuvel, S.* 161
Methodology in the Study of Attitudes of Africans. *Biesheuvel, S.* 169
The Psycho-Motor Development of African Children in the First Year, and the Influence of Maternal Behavior. *Geber, M.* 185
Child Animism: I. A Critical Survey of Cross-Cultural Research. *Jahoda, G.* 197
Child Animism: II. A Study in West Africa. *Jahoda, G.* 213
Some Results of an Enquiry into the Influence of Child-Training Practices on the Development of Personality in a Bacongo Society (Belgian Congo). *Knapen, M. T.* 223
Children's Earliest Conceptions of Economic Relationships (Australia). *Danziger, K.* 231
Immanent Justice Among West African Children. *Jahoda, G.* 241
Some Methodological Considerations Concerning the Study of Aptitudes and the Elaboration of Psychological Tests for African Natives. *Verhaegen, P., and Laroche, J. L.* 249
Acculturation of Attitude and Personality Among Katangese Women. *Leblanc, M.* 257
Social Influences in Zulu Dreaming. *Lee, S. G.* 265
The Bantu Clerk: A Study of Role Expectations. *Sherwood, R.* 285
Self Interpretations of Group Differences in Values (Natal, South Africa). *Danziger, K.* 317
Speed and Accuracy of Performance of an African Native Population of Belgian Children on a Paper-and-Pencil Percept Task. *Ombredane, A., et al.* 327
The Impact of Education on Non-Scientific Beliefs in Ethiopia. *Lord, E.* 339
A Comparison of Test Scores for the 16-17-year Age Group of Navaho Indians with Standardized Norms for the Wechsler Adult Intelligence Scale (Arizona and New Mexico). *Howell, R. J., et al.* 355
A Comparison of Attitude Scores of Some Politico-Economic Issues Between Two Samples of College Students in Orissa, India, After an Interval of Four Years. *Rath, R.* 361
A Study in Stereotypes of College Freshmen and Service Holders in Orissa, India, Towards Themselves and Four Other Foreign Nationalities. *Rath, R., and Das, J. P.* 373
National Stereotypes of University Students in East Pakistan. *Zaidi, S. M., and Ahmed, M.* 387
Is the Tolerant Personality Type the Opposite of the Intolerant? (Western Australia). *Taft, R.* 397

VOL. 48 AUGUST, 1958

Petition-Signing as Adjustment to Situational and Personal Factors. *Helson, H., et al.* 3
Social Expectancy as a Function of Judged Social Distance. *Muraskin, J., and Iverson, M. A.* 11

Prejudicial Attitudes and the Identification of Their Stimulus Objects: A Phenomenological Approach. *Cooper, J. B.* 15
Sex Differences in Self Assessment. *Talland, G. A.* 25
Some Effects of Communication Requirements in Group Structures. *Shelly, M. W., and Gilchrist, J. C.* 37
A Factor Analysis of Self-Ratings on Social Behavior. *Storm, T., et al.* 45
An Experimental Investigation of the Effectiveness of the "Big Lie" in Shifting Attitudes. *Helson, H., et al.* 51
Hobbes Revisited: An Analysis of the Compatibility of the Theories of Human Nature and of the State in the *Leviathan. Stauffer, R. B., and Vinacke, W. E.* 61
An Investigation of Anti-Semitic Feelings in Two Groups of College Students: Jewish and Non-Jewish. *Engel, G., et al.* 75
Religious Values and Authoritarian Tendency. *Jones, M. B.* 83
The Logarithmic Relation of Social Distance and Intensity. *Dodd, S. C., and Griffiths, K. S.* 91
Differential Adjustment to Internment Camp Life. *Tagiuri, R.* 103
Progressivism and Traditionalism: Basic Factors of Educational Attitudes. *Kerlinger, F. N.* 111
The Influence of Racial Group Composition on Sociometric Choices and Perceptions. *Mann, J. H.* 137
Value Conformity in Normal and Non-Normal Groups. *Holder, W. B.* 147

VOL. 22 J. SPEECH HEAR. DIS. DECEMBER, 1957

Relation Between EEG Pattern and Ease of Eliciting Electrodermal Responses. *Charan, K. K., and Goldstein, R.* 651
What is Zero Hearing Loss? *Davis, H., and Usher, J. R.* 662
Children as Guides to Teaching. *Beasley, J. E.* 691
Suggestions for Terms Applied to Electrophysiologic Tests of Hearing. *Goldstein, R., and Derbyshire, A. J.* 696
Four Psychological Scaling Methods Applied to Articulation Defectiveness. *Sherman, D., and Moodie, C. E.* 698
Behavioral Rigidity in Speech-Handicapped Children. *Kapos, E., and Fattu, N. A.* .. 707
Stuttering as Conflict: 1. Comparison of Therapy Techniques Involving Approach and Avoidance. *Sheehan, J. G., and Voas, R. B.* 714
The Frequency and Duration of Treatment Sessions in Speech Correction. *McDonald, E. T., and Frick, J. V.* 724
Problems in Military Audiometry: A CHABA Symposium. 1. Introduction to the Presentations. *Usher, J. R.* 729
Problems in Military Audiometry: A CHABA Symposium. 2. Veteran's Compensations for Hearing Loss. *Johnson, K. O.* 731
Problems in Military Audiometry: A CHABA Symposium. 3. Diagnostic Audiometry. *Hoople, G.* 734
Problems in Military Audiometry: A CHABA Symposium. 4. A Classification of Hearing Tests. *Hirsh, I. J.* 736
Problems in Military Audiometry: A CHABA Symposium. 5. Review of the "Report by the Working Group on Military Audiometry". *Carhart, R.* 744
Problems in Military Audiometry: A CHABA Symposium. 6. Automatic Audiometry. *Webster, J. C.* 748
Problems in Military Audiometry: A CHABA Symposium. 7. Practical Limitations in Military Audiometry. *Glorig, A.* 750
Problems in Military Audiometry: A CHABA Symposium. 8. Audiometry in the Armed Forces. *The Chaba Council* 752
Noise Bands *vs.* Pure Tones as Stimuli for Audiometry. *Myers, C. K.* 757
The C. S. Bluemel Collection on Stuttering. *Murray, E.* 761
Use of Palatography. *Ladefoged, P.* 764

VOL. 8 NEUROL. APRIL, 1958

Introduction. *Forster, F. M.* 1
Origins and Development of Neurochemistry. *Tower, D. B.* 3
Maintenance of the Composition of Isolated Cerebral Tissues. *McIlwain, H.* 32
Metabolism of Nucleic Acids and Lipids in Brain and in Peripheral Nerve During Experimental Demyelination. *Rossiter, R. J., et al.* 34
A Specific Antidote for Nerve Gas and Insecticide (Alkylphosphate) Intoxication. *Wilson, I. B.* 41
*Some Aspects of the Relation of Ceruloplasmin to Wilson's Disease. *Scheinberg, I. H., et al.* 44
Muscle Proteins in Dystrophy. *Horvath, B.* 52
A Collagen-like Compound Isolated from Spinal Cord. *Roboz, E., et al.* 54
Nucleic Acid Content of Cerebral Neurons. *Grenell, R. G., and May, L.* 56
Quantitative Histochemistry of Selected Central Tracts. *McDougal, D. B.* 58

Substrate Utilization of Peripheral Nerves. *Heller, I. H., and Hesse, S.* 60
Increase in Rate of Glucose Utilization During Activity in Excised Sympathetic Ganglia. *Dolivo, M., and Larrabee, M. G.* 62
Effect of Glutamate and Glutamine on the Intracellular Electrolytes of Nervous Tissue. *Ames, A., III* 64
Diffusion Constants in the Extracellular Space of Brain Tissue. *McLennan, H.* 67
Mitochondria in Brain as the Sites of its Metabolic Activity. *du Buy, H. G.* 69
The Adenosine Polyphosphates, Creatine Phosphate and Ascorbic Acid Content of Rat Brain. *Lin, S., et al.* 72
Some Aspects of Oxidative Metabolism of Glucose in Brain Suspensions. *Gordon, M., et al.* 75
Metabolic Studies of Gamma-Aminobutyric Acid. *Roberts, E., and Baxter, C. F.* 77
Studies with Tritium-Labeled Inositol. *Agranoff, B. W., and Brady, R. O.* 79
Studies on the Biosynthesis of Cerebroside Galactose. *Moser, H., and Karnovsky, M. L.* 81
Studies on the Biosynthesis of Galactolipids. *Burton, R. M., et al.* 84
Biochemical Comparison of Wallerian Degeneration in the Central and Peripheral Nervous Systems. *McCaman, R. E., and Robins, E.* 86
Studies on Myelination and Demyelination: Lipogenesis by Nervous Tissue *in Vitro*. *Majno, G., and Karnovsky, M. L.* 88
Mode of Action of an Inhibitor of Acetylcholine Synthesis. *MacIntosh, F. C., et al.* 90
Correlation of Rate of Compulsive Circling in Rabbits with Asymmetric Accumulation of Acetylcholine in Cerebral Cortices. *Aprison, M. H.* 92
Butyrylcholinesterase in Human and Ruminant Nervous Tissue. *Bernsohn, J., and Possley, L. R.* 94
*Effect of Prolonged Treatment with Tranquilizing Drugs upon Two Enzymes of the Central Nervous System. *Ashby, W.* 96
Factor I and Gamma-Aminobutyric Acid: A Summary. *Elliott, K. A. C.* 98
Cerebral Blood Flow in Epileptic Children During the Interseizure Period. *Kennedy, C., et al.* 100
N-Methylated Derivatives of Barbituric Acid, Hydantoin and Oxazolidinedione Used in the Treatment of Epilepsy. *Butler, T. C., Waddell, W. J.* 106
Relation Between Excitability and Metabolism in Brain as Elucidated by Anticonvulsant Drugs. *Woodbury, D. M., et al.* 113
Sound-Induced Seizures in Animals. *Suter, C., et al.* 117
Sound-Induced Seizures in Animals: The Efficiency of Certain Anticonvulsants in Controlling Sound-Induced Seizures in DBA/2 Mice. *Suter, C., et al.* 121
Sound-Induced Seizures in Animals: Magnesium Deficiency and Sound-Induced Seizures in Rats. *Suter, C., et al.* 125
Composition of Cerebrospinal Fluid under Varying Conditions. *Bering, E. A.* 129

Some Aspects of the Relation of Ceruloplasmin to Wilson's Disease

The results of determinations of the concentration of the plasma protein, ceruloplasmin, in various clinical conditions are presented. These show that Wilson's disease is characterized by a marked deficiency or absence of the protein, but that neither liver disease nor neurologic disease of various other kinds is associated with diminished concentrations of ceruloplasmin. In fact, in both of these latter groups the mean value for plasma ceruloplasmin was significantly ($P < 0 \cdot 01$) greater than the mean of the normal group. Although the mean concentration of plasma ceruloplasmin in a group of schizophrenic patients was slightly higher than the mean of the normal group, determination of the concentration of ceruloplasmin seems to be of no value in differentiating schizophrenic from normal individuals. Studies performed in patients with Wilson's disease indicate that their deficiency of ceruloplasmin is a consequence of impaired synthesis of the protein.

(Authors' Abstr.)

Effect of Prolonged Treatment with Tranquilizing Drugs upon Two Enzymes of the Central Nervous System

Superimposed upon a fluid state of carbonic anhydrase content of the central nervous system in the controls, the data presented would seem to clearly indicate that the tranquilizing drugs produce an increase in carbonic anhydrase in areas of the brain, which appears to culminate in the hippocampus and brainstem. In growing animals the effect is more diffuse than in adult animals. There has been shown an optimum point in the course of injections after which the carbonic anhydrase content deteriorates.

(Author's Abstr.)

APRIL

The Neuropathies of Diabetes. *Sullivan, J. F.* 243
*Uncinate Fits. *Daly, D.* 250
Unusual Form of Cerebellar Ataxia with Sex-Linked Inheritance. *Malamud, N., and Cohen, P.* 261
Effect of Methylphenidate Hydrochloride in Antagonizing Barbiturate-Induced Depressions. *Ticktin, H., et al.* 267

Distribution of Cutaneous Hyperalgesia. *Klingon, G. H., and Jeffreys, W. H.* 272
Cerebral Hemispheric Tumors and Extrapyramidal Signs and Symptoms. *Roth, R. L., and Bebin, J.* 277
Progressive Demyelinating Leukoencephalopathy. *Wohlwill, F. J., and Paine, R. S.* 285
Demonstration of Carotid Occlusion by Small Dose Arteriography. *Oldendorf, W. H.* 296
Free Amino Acid Content of Cerebrospinal Fluid in Humans and Dogs. *Logothetis, J.* 299

Uncinate Fits

1. The history of the concept of uncinate seizures is reviewed. Observations on 55 patients form the basis of this report.

2. Olfactory phenomena occurred most frequently, usually in the form of hallucinations. The hallucinations ranged from crude, unrecognizable sensations of smell to complex multisensorial elaborations. In rare instances olfactory "reminiscences" and illusions are reported.

3. The uncus appears to be the cortical centre for smell. In one patient necropsy revealed focal gliosis in the uncus.

4. Gustatory phenomena were limited to undifferentiated hallucinations of taste.

5. Twelve patients reported rhinostomal sensations consisting of burning, tingling, or crawling sensations in the nose, mouth, or throat. In the majority they were associated with olfactory or gustatory hallucinations.

6. Interictal or postictal illusions of smell or taste or, less commonly, hallucinations, are described by some patients.

7. The relationship of these phenomena to "dreamy states", ictal affect, and vegetative disturbances during seizures was analysed. An attempt was made to extend the concept of the functions of this area to explain the association of these events.

(Author's Abstr.)

MAY

*The Use of Anticoagulants in Cerebral Thrombosis. *Fisher, C. M.* 311
The Electroencephalogram in Metastatic Tumors of the Brain. *Klass, D. W., and Bickford, R. G.* 333
What Neurologists Should Know About International Neurologic Congresses. *Van Bogaert, L., and Radermecker, J.* 338
Production of Basal Ganglia Lesions by Chemopallidectomy. *Cooper, I. S., and Bravo, G. J.* 344
Paresis and Hyperexcitability in Adynamia Episodica Hereditaria. *Buchthal, F., et al.* 347
Effects of Secondary Pallidal and Striatal Lesions upon Cerebellar Dyskinesia. *Carpenter, M. B., et al.* 352
Distribution of High Energy Phosphate in Normal and Dystrophic Muscle. *Ronzoni, E., et al.* 359
Neurologic Implications of Behcet's Syndrome. *Whitty, C. W. M.* 369
Cerebrospinal Fluid Free Amino Acids in Neurologic Diseases: Observations in Aspartic Acid Concentrations. *Logothetis, J.* 374
A Classification and Outline of Cerebrovascular Diseases 395

The Use of Anticoagulants in Cerebral Thrombosis

Anticoagulant drugs have been administered to 58 patients with symptoms and signs of the various stages of cerebral thrombosis due to atherosclerosis. The data suggest that anticoagulant therapy abolishes transient ischemic attacks and prevents or postpones the arrival of a threatening stroke. When used in the early stage of a progressively evolving stroke due to cerebral thrombosis, the downhill course appears to be satisfactorily arrested and reversed. Patients observed for periods both on and off anticoagulants fared better while receiving anticoagulants. A group of 37 cases rather comparable to the anticoagulant group were observed without anticoagulant therapy and ran a far less satisfactory course than the treated patients. From this preliminary clinical exploration, anticoagulant therapy may be regarded as a promising therapy in cases of cerebral thrombosis encountered in the earlier stages; certainly in the author's experience it seems more beneficial than any other mode of therapy used at present and it is relatively safe. The increasing frequency with which stroke patients are seen in the clinic, at a point in their illness when anticoagulants might be effective, indicates that this therapy may be of great significance in medical practice. The present data do not provide incontrovertible evidence of the therapeutic efficacy of anticoagulants because satisfactory control patients have not been studied.

The tentative indications for anticoagulant therapy in transient cerebral ischemic attacks and cerebral thrombosis have been discussed at some length.

The clinical picture of transient ischemic attacks has been described and their differential diagnosis discussed.

(Author's Abstr.)

JUNE

Focal Seizures Due to Chronic Localized Encephalitis. *Rasmussen, T., et al.* 435
Comparative Effects of Relaxant Drugs on Human Skeletal Muscle Hyperactivity. *Vazuka, F. A.* 446

Spinal Fluid Protein in Tumors of the Sellar Region. *Bakay, L.*	455
Alcoholic Gasserian Injection for Relief of Tic Douloureux: Preliminary Report of a Modification of Penman's Method. *Ecker, A., and Perl, T.*	461
Relationship of Dystrophia Myotonica (Myotonic Dystrophy) and Myotonia Congenita (Thomsen's Disease). *Caughey, J. E.*	469
*Localization Within the Amygdaloid Complex of Anesthetized Animals. *Wood, C. D., et al.*	477
Vertebral Deformities and Other Abnormalities in Charcot-Marie-Tooth Disease. *Smith, C. K.*	481
*The Narcolepsy Syndrome. *Ganado, W.*	487
*Subcutaneous Hemorrhages in Multiple Sclerosis. *Swank, R. L.*	497

Localization Within the Amygdaloid Complex of Anesthetized Animals

1. The corticomedial division of the amygdaloid complex appears to be the area concerned with regulation of autonomic responses from this region.
2. The blood pressure responses from the amygdaloid area appear to be mediated through the region of the ventromedial nucleus of the hypothalamus. Respiratory responses from the amygdaloid complex also appear to be associated with this structure.
3. The basolateral division appears to be concerned with the somatic responses reported from this area, especially with movements of the face and jaw. Sustained jaw opening was obtained at lowest threshold from the basal medial nucleus, and the lowest threshold for jaw closure was found to be in the lateral nucleus.
4. The reactions elicited by stimulation of the amygdaloid complex appear to be a function of this area and not due to spread of stimulation current to adjacent structures.

(Authors' Abstr.)

The Narcolepsy Syndrome

A series of 158 cases with narcolepsy has been reviewed.

Evidence is strongly suggestive that the narcolepsy syndrome is a nonepileptic disturbance of subcortical origin. It appears to be a state of depression of the arousal system, possibly from a neurohumoral deficiency.

(Author's Abstr.)

Subcutaneous Hemorrhages in Multiple Sclerosis

Frequent and periodic occurrence of small spontaneous subcutaneous hemorrhages in multiple sclerosis is reported.

(Author's Abstr.)

JULY

Peripheral Neuropathy in Multiple Sclerosis. *Hasson, J., et al.*	503
Neurologic Manifestations of Myxedema. *Nickel, S. N., and Frame, B.*	511
Residual Function after Subtotal Spinal Cord Transection in Adult Cats. *Windle, W. F., et al.*	518
Effect of Acetazolamide on CSF Pressure and Electrolytes in Hydrocephalus. *Birzis, L., et al.*	522
Clinical Evaluation of Spirodon. *Frank, G., and Morrell, F.*	529
Acute Cerebellar Syndrome of Childhood. *Blaw, M. E., and Sheehan, J. C.*	538
*Investigation of the Primate Amygdala: Movements of the Face and Jaws After-discharge and the Anterior Commissure. *Frost, L. L., et al.*	543
Regulation of Carbohydrate Metabolism in Spinal Cord Injuries. *Brown, M., et al.*	547
Platelet Agglutinins in the Spinal Fluid in Multiple Sclerosis. *Kittleson, K. D., and Alt, H. L.*	551
Is Needle Puncture of the Brain Entirely Harmless? *Dekaban, A. S.*	556
Cerebral Hemodynamics and Metabolism in Mental Deficiency. *Fazekas, J. F., et al.*	558
*Somatic and Visceral Responses from the Cingulate Gyrus. *Showers, M. J. C., and Crosby, E. C.*	561
Cell Count in the Lateral Geniculate Nucleus of Man. *Sullivan, P. R., et al.*	566

Investigation of the Primate Amygdala: Movements of the Face and Jaws Afterdischarge and the Anterior Commissure

Gloor has summarized the evidence regarding the known corticopedal pathways of the amygdaloid complex. Such pathways go to the homologues of the temporal polar and insular cortex. In this investigation an after-discharge was recorded from the parietal cortex of the monkey following repetitive stimulation of the homolateral amygdaloid complex. The latency of this response was not measured, but such an after-discharge may indicate a polysynaptic pathway between the amygdaloid complex and the homolateral parietal cortex.

Bucy and Kluver, Lauer, and others have found that a part of the anterior commissure provides an anatomic connection between the amygdaloid complexes. It has been demonstrated here that a repetitive electrical stimulus in one amygdaloid complex can be followed by an afterdischarge in the other amygdaloid complex and this can be recorded from the region of the anterior commissure. When the afterdischarge was recorded from the amygdaloid complex, the facial movements which had been ipsilateral to the side of the stimulation became bilateral. After the anterior commissure was severed, it was no longer possible to record the after-discharge in the amygdaloid nucleus contralateral to the one stimulated, and the facial movements pursuant to stimulation of one amygdaloid complex remained ipsilateral to the side stimulated.

These experiments have demonstrated that epileptiform discharges may arise in the amygdaloid complex of one temporal lobe and spread to the amygdaloid area of the other temporal lobe by way of the anterior commissure, and that such spread can be prevented by interrupting the anterior commissure.

(Authors' Abstr.)

Somatic and Visceral Responses from the Cingulate Gyrus

1. An account is presented of the results of stimulation and destruction of portions of the cingulate region of 15 Macaca mulatta monkeys. A pattern of somatic movements was found on the anterior portion of the cingulate gyrus which repeated itself in reverse order on the posterior portion of this gyrus. In this pattern, movements characteristic of emotional expression, involving the musculature of the face, the head, and the neck were present upon stimulation of all points on the gyrus. They were either homolateral or bilateral. The movements involving clonic flexion, extension, and internal rotation of the upper and lower extremities were either contralateral or bilateral.
2. Increased strength of stimulation (7 to 10 volts) produced convulsive movements comparable to those seen in certain forms of epilepsy.
3. Autonomic phenomena included respiratory, cardiovascular, sudoriferous and pupillary changes, and piloerection. These resulted both from stimulation and from destruction of the rostral and the caudal parts of the cingulate gyrus.

(Authors' Abstr.)

AUGUST

Studies on the "Cerebral Pacemaker". *Aird, R. B., and Garoutte, B.*	581
Promising Results in Cryptococcal Meningitis. *Rubin, H., and Furcolow, M. L.*	590
Intracranial Pneumoplanigraphy in the Verticosubmental Position. A Neuroroentgenographic Refinement. *Stern, W. E., et al.*	596
Localized Cerebral Hypoglycemia Simulating Stroke. A Clinical and Experimental Study. *Meyer, J. S., and Portnoy, H. D.*	601
*Epileptic Cephalea. *Halpern, L., and Bental, E.*	615
Therapy of Acute, Chronic and Mixed Hepatic Porphyria Patients with Chelating Agents. *Peters, H. A., et al.*	621
Treatment of Heredodegenerative Disorders. A Continuing Challenge to Neurology. *Klingman, W. O.*	633
Excitability in Experimental Myopathy. 1. Measurement of Refractory Period; Quinidine Effect; Cortisone Myopathy. *Glaser, G. H., and Stark, L.*	640
Observations on Hemiplegia with Middle Cerebral Artery Trunk Occlusions and with "Normal" Carotid Angiograms. *Webster, J. E., and Gurdjian, E. S.*	645

Epileptic Cephalea

Five cases of paroxysmal headaches in children are described.

The nature of the headaches was clarified by electroencephalograms, which revealed epileptic activity in all cases, especially during photic stimulation.

With the exception of one who had convulsions in the first week of her life, none of these children had experienced frank seizures.

Epileptic cephalea seems to appear as the sole manifestation of a latent epileptic tendency and deserves a special place among the various forms of headaches.

(Authors' Abstr.)

SEPTEMBER

Cluster Headaches. *Friedman, A. P., and Mikropoulos, H. E.*	653
The Prognosis of Some Brain Stem Vascular Syndromes. *Currier, R. D., et al.*	664
Relapsing Inclusion Encephalitis (Dawson Type) of Eight Years' Duration. *Landau, W. M., and Luse, S. A.*	669
Progressive Cerebral Degenerations of Childhood. *Stevens, H., and Dekaban, A.*	677
Clinical and Electroencephalographic Effects of Megimide in Patients Without Cerebral Disease. *Green, M. A., and Fink, M.*	682
Tilt Table Electroencephalography in Insufficiency Syndromes. *Weiss, S., and Froehlich, W.*	686
Effects of Bilateral Cerebral Infarction on Respiratory Center Sensitivity. *Heyman, A., et al.*	694
*Implications of a Five-Year Study of 700 Basal Ganglia Operations. *Cooper, I. S., and Bravo, G. J.*	701
Excitability in Experimental Myopathy. II. Potassium Deficiency: An Initial Study. *Glaser, G. H., and Stark, L.*	708

Neurologic Complications in Patients with Coarctation of the Aorta. *Tyler, H. R., and Clark, D. B.*	712
A Simple Device for Measuring the Visual Motor Reaction Time. *Schwab, R. S.*	719

Implications of a Five-Year Study of 700 Basal Ganglia Operations

A five-year follow-up study of more than 700 operations of anterior choroidal occlusion, chemopallidectomy, and chemothalamectomy leads to the following conclusions:

1. This investigation has resulted in a roentgen-anatomic-physiologic method of basal ganglia surgery which can relieve tremor and rigidity of Parkinsonism in 80 per cent. of properly selected cases, with a risk of mortality of 2·4 per cent. and a risk of hemiparesis of 3 per cent.

2. Tremor, rigidity, deformity, and incapacitation of parkinsonism can be relieved without the infliction of any neurologic or psychologic deficit.

3. Five-year cures of the symptoms of tremor, rigidity, deformity, and incapacitation can now be documented in this series of cases.

4. On the basis of this study, parkinsonism can no longer be considered a hopeless, progressive, incurable disease. Inasmuch as the incapacitating symptoms of tremor, rigidity, and deformity have been alleviated by surgery of the globus pallidus and thalamus for as long as five years without recurrence, a more hopeful and realistically optimistic viewpoint can be adopted concerning this disease. The therapeutic implications of this statement apply not only to surgical therapies but to the future development of medical treatment as well. Moreover, the symptoms of dystonia musculorum deformans have been alleviated during the course of this study for periods up to three years without recurrence. Thus, this disease can no longer be considered as necessarily incurable or irreversibly progressive.

5. The treatment of choice of many cases of parkinsonism and of all cases of dystonia musculorum deformans is surgery of the globus pallidus and thalamus.

6. Lesions confined to the globus pallidus and/or ventrolateral region of the thalamus on one side of the brain do not result in any motor, sensory, intellectual, or psychologic deficit. The extent to which such lesions can be inflicted on both sides of the brain requires further elucidation.

7. Clinical studies leading to a more physiologic classification of the subdivisions of the overall parkinsonism syndrome are required if therapy and further basic investigations are to be conducted intelligently and fruitfully.

8. Many of the older concepts of basal ganglia function, pathogenesis of tremor and rigidity, and therapy of hyperkinetic diseases have been rendered suspect and some completely disproved by this and other investigations. There is a further need for evaluation of many concepts of extrapyramidal disease which have been allowed to become dogmatic and which do not occur with existing facts. However, not only must we be willing to re-evaluate former concepts, but we must also be on guard against replacing them with new dogmas too soon. Our entire understanding of the role of basal ganglia and the extra-pyramidal system must be viewed in the context of the contributions of these structures to overall brain function. Our present state of knowledge does not permit more than working hypotheses. These should not be misinterpreted as facts until they are proved beyond any doubt.

(Authors' Abstr.)

OCTOBER

Skeletal Abnormalities with Motor System Disease. *Kurland, L. T., and Myrianthopoulos, N. C.*	727
*Long-Range Effects of Electropallidoansotomy in Extrapyramidal and Convulsive Disorders. *Spiegel, E. A., et al.*	734
Electrooculographic Investigation of the Caloric Response. *Anderson, P. J., et al.*	741
Impairment of Flicker Discrimination in Brain-Damaged Patients. *Parsons, O. A., and Huse, M. M.*	750
Multiple Sclerosis and Allied Diseases in Japan. *Okinaka, S., et al.*	756
Critical Flicker Frequency and EEG Findings in Patients with Brain Damage. *Kooi, K. A., et al.*	764
*A New Drug for Petit Mal Epilepsy. *Zimmerman, F. T., and Burgemeister, B. B.*	769
The Fate of Offspring from Mothers Receiving Sodium Pentobarbital Before Delivery. *Becker, R. F., et al.*	776
Pneumotaxic Localization, Recording, Stimulation, and Section of Basal Brain Structures in Dyskinesia. *Bertrand, C., et al.*	783
Effects of Blood *p*H and Carbon Dioxide on Cerebral Electrical Activity. *Swanson, A. G., et al.*	787
Biothesiometry in the Diagnosis of Lumbar Disk Protrusion. *Steiness, I.*	793
Neurology at the Crossroads. *Walshe, F.*	796

Long-range Effects of Electropallidoansotomy in Extrapyramidal and Convulsive Disorders

Follow-up studies of stereotaxic operations on the basal ganglia performed from 1948 to 1958 are presented.

Fractionated electropallidoansotomy was applied to (1) paralysis agitans and parkinsonism, (2) other involuntary movements of extrapyramidal origin, and (3) certain types of spasticity, including a case of tonic perseveration of voluntary innervation. In a fourth group—convulsive disorders refractory to anticonvulsive medication and showing seizure discharges in the forebrain ganglia—pallidoansotomy was used alone or combined with amygdalotomy.

(Authors' Abstr.)

A New Drug for Petit Mal Epilepsy

This article reports upon the effect of PM 671 in controlling or reducing petit mal attacks in 109 patients, many of whom were considered intractable cases.

The present drug, PM 671, on the basis of effectiveness against petit mal attacks, mildness of side effects, and holding ability, offers more promise than other succinimide compounds and medications tested in the past ten years.

Quick and dramatic reduction of attacks occurred in most of 109 patients studied, and side effects were mild and infrequent. Side reactions were occasional drowsiness and dizziness, with some nausea and gastric distress—usually on high doses. Nausea and gastric distress are basic side effects to all succinimide compounds, but, fortunately, they are rare and not particularly disturbing. No deleterious changes in the blood picture have been noted to date, and urinary findings are within normal limits. No skin rashes have been noted.

Studies revealed that PM 671 is also outstanding in the lack of "slippage"—that property in a drug of slowly losing its holding power. Quite a few patients, never before helped by any drug, have been completely controlled for several years on PM 671.

For the group as a whole, an overall 85 per cent. reduction in attacks was obtained in the treatment of petit mal epilepsy. Complete control of petit mal attacks was secured in 42 per cent. of patients treated; practical control with an 80 to 99 per cent. reduction in attacks in 24 per cent.; and partial control, 5 to 79 per cent. reduction, in 26 per cent.

Experience to date indicates that PM 671 is essentially a petit mal drug. Much more effectiveness was noted in the treatment of pure petit mal cases—where pure petit mal existed alone or was combined with other types of seizures—than when a "mixed" petit mal element was present. Results showed that 83 per cent. of all pure petit mal cases had a reduction in attacks of at least 80 per cent. after therapy; only 29·5 per cent. of the mixed petit mal cases reached this high level of control. The individual frequency of attacks likewise did not appear to have any relation to the effectiveness of medication.

(Authors' Abstr.)

VOL. 71 **PISANI.** SEPTEMBER-DECEMBER, 1957

Research on the Children's Apperception Test in Epileptic Children, etc. *Biondi, L.*	409
Consideration on the Long Term Results of Treating Mental Illness with Serpasil. *Santangelo, G.*	419
Schizophrenia With a Common Nosology. *Dalma, G.*	429
A Case of Paranoia Treated by Psychodrama. *Moreno, J. L.*	447

VOL. 72 JANUARY-APRIL, 1958

Clinical Considerations of the First Results of Treating a Group, Predominantly Schizophrenic, with Thiazine Derivatives and a Barbiturate. *Maccaqnani, G.*	3
Neurological and Psychological Aspects of Problems in Immature Children. *Circo, P.*	25
Syndrome of Spinal Lesions Arising During Cooley's Disease. *Taibi, G.*	47
A Contribution to the Study of Exogenous Toxic Psychoses. *Vitagliano, G.*	57
Tissue Treatment in Poliomyelitis. *La Loggia, M.*	65
A Clinical Contribution to the Study of Geriatric Problems. *Bisio, B.*	73
The Psychopathological Evolution of Patients Seven Years after Leucotomy. *Binenti, B.*	119
Thrombosis of the Carotid Following Arteriography. *Beluffi, M., et al.*	127
A Report on Dimethylaminopropylphenothiazine in Psychiatric Treatment. *Bisio, B.*	145

VOL. 21 **PSYCHIAT.** AUGUST, 1958

Intuition in the Methodology of Psychoanalysis. *Board, R.*	233
Problems of Communication Between Doctor and Patient in Psychotherapy. *Weigert, E.*	241
Language of the Dyad. *Jaffe, J.*	249
Fostering the Involvement of the Psychiatric Patient in Group Activities. *Goodrich, D. W., et al.*	259
The Group Focal Conflict. *Whitman, R. M., and Stock, D.*	269
Family Interaction in the Therapy of Adolescent Patients. *Miller, D. H.*	277
Preanalytic Preparation for the Therapeutic Process in Schizophrenia. *Hoedemaker, E. D.*	285

VOL. 27 **PSYCHO-ANAL. QUART.** APRIL, 1958

1879-1958. *Jones, E.*	157
Dreams of the Blind. *Blank, H. R.*	158
A Contribution to the Theory of Play. *Alexander, F.*	175
Notes on a Case of *Maladie des Tics. Aarons, Z. A.*	194
Blanket Interpretations. *Feldman, S. S.*	205
Some Affective Meanings of Dizziness. *Giovacchini, P. L.*	217

Research Into the Process of Supervision in Psychoanalysis. *Kubie, L. S.* 226
An Approach to a Systematic Study of Ego Function. *Sheppard, E., and Saul, L. J.* .. 237
A Special Mechanism of Pathological Weeping. *Lacombe, P.* 246

JULY

Models for Pleasure. *Buchenholz, B.* 307
The Fantasy of Being Rescued in Suicide. *Jensen, V. W., and Petty, T. A.* 327
Narcissistic Defenses During Pregnancy. *Fox, H. M.* 340
The Impostor. *Greenacre, P.* 359
Underlying Ego Mechanisms in Delinquency. *Bernabeu, E. P.* 383
"When I Grow Big and You Grow Little." *Jarvis, W.* 397
The Genesis of Moses. *Slap, J. W.* 400
Footnote to the Genesis of Moses. *Naftalin, M.* 403
1909-1958. *Fabian, A.* 406

VOL. 20 PSYCHOSOM. MED. MARCH-APRIL, 1958

Psychoanalytic Contributions to Psychosomatic Research. *Linn, L.* 88
Shifts Along the Alert-Repose Continuum During Remission of Catatonic "Stupor" with Amobarbital. *Stevens, J. M., and Derbyshire, A. J.* 99
Treatment of Therapeutic Blockades with Thiopental (Pentothal) Sodium and Methamphetamine (Desoxyn), 1948-1957. *Scarbrough, H. E., and Wheelis, D. B.* 108
Effect of Psychoanalysis on the Course of Peptic Ulcer. *Orgel, S. Z.* 117
Psychological Factors and Reticuloendothelial Disease: IV. Observations on a Group of Children and Adolescents with Leukemia; An Interpretation of Disease Development in Terms of the Mother-Child Unit. *Greene, W. A., Jr., and Miller, G.* 124
Transvestism and Pruritus Perinei. *Greenberg, N. H., and Rosenwald, A. K.* 145
A Case of Schizophrenia in Which Manifestations of Parkinsonism Appeared During the Course of the Psychosis and Disappeared After Lobotomy. *Guggenheim, P., and Cohen, L. B.* 151

JULY-AUGUST

Relationship of Separation and Depression to Disease: I. A Report on a Hospitalized Medical Population. *Schmale, A. H., Jr.* 259
An Investigation of the Relation Between Life Experience, Personality Characteristics, and General Susceptibility to Illness. *Hinkle, L. E., Jr., et al.* 278
Masochism and Interstitial Cystitis: Report of a Case. *Bowers, J. E., et al.* 296
Observations on Blood Pressure and Other Physiologic and Biochemical Mechanisms in Rats with Behavioral Disturbances. *Shapiro, A. P., and Melhado, J.* 303
Modifications of the Oropharangeal Bacteria with Changes in the Psychodynamic State: II. A Validation Study. *Kaplan, S. M., and Gottschalk, L. A.* 314
Effect of Hypnosis on Intracellular Pressure in Normal and Glaucomatous Subjects. *Berger, A. S., and Simel, P. J.* 321

VOL. 18 QUART. J. STUD. ALC. DECEMBER, 1957

*Partial Equivalence of Chronic Alcohol and Barbiturate Intoxications. *Fraser, H. F., et al.* 541
Dietary Deficiency in Mice in Relation to Voluntary Alcohol Consumption. *Mirone, L.* 552
A Study of Addiction to Nonethyl Alcohols and Other Poisonous Compounds. *Mendelson, J., et al.* 561
Glutamine in the Treatment of Alcoholism. A Preliminary Report. *Rogers, L. L., and Pelton, R. B.* 581
Alcoholism in Women: Social and Psychological Concomitants. I. Social History Data. *Lisansky, E. S.* 588
A Follow-Up Study of Treated Alcoholics. *Pfeffer, A. Z., and Berger, S.* 624
The Development of a Research Methodology for Achieving the Cooperation of Alcoholics and Their Nonalcoholic Wives. *Mitchell, H. E., and Mudd, E. H.* .. 649
Integration of Alcoholism and Mental Health Services. *McGee, W. R., and Scher, S. C.* 658
Disulfiram as an Aid to Psychotherapy in the Case of an Impulsive Drinker. *Winship, G. M.* 666

Partial Equivalence of Chronic Alcohol and Barbiturate Intoxications

1. Ten morphine addicts in good physical health, eight of whom were addicted to barbiturates, volunteered for an experimental study which involved withdrawal of opiates, continuous intoxication with pentobarbital or secobarbital for 22 to 44 days, substitution of alcohol for barbiturates for 14 days and, finally, abrupt withdrawal of alcohol.

2. The clinical symptoms of the intoxications induced by barbiturates and by alcohol were similar, but after substitution of alcohol the EEG pattern changed from that characteristic of barbiturates to that characteristic of alcohol.

3. The "major" symptoms of abstinence due to abrupt withdrawal of barbiturates are convulsions and delirium. The anticipated frequency and severity of these symptoms were reduced significantly by the substitution of alcohol. The incidence and severity of "minor"

symptoms of barbiturate abstinence (anxiety, insomnia, tremor, anorexia, vomiting and weight loss) were reduced.

4. When alcohol was discontinued abruptly after 14 days of substitution, convulsions or delirium or both appeared in three of nine patients who completed the experiment.

5. It is concluded that alcohol is not a complete substitute for barbiturates in patients chronically intoxicated by pentobarbital or secobarbital, but that it is a partial substitute.

(Authors' Abstr.)

VOL. 19 JUNE, 1958

Studies on Severe Alcohol Intoxication in Dogs. I. Blood and Urinary Changes in Lethal Intoxication. *Klingman, G. I., and Haag, H. B.* 203
Alcohol as a Depressant in Psychological Conflict in Rats. *Weiss, M.* 226
A Controlled Trial of Chlorpromazine and Promazine in the Management of Delirium Tremens. *Laties, V. G., et al.* 238
Reference Group Influences on Student Drinking Behavior. *Rogers, E. M.* 244
Criminal Careers of the Chronic Police Case Inebriate. *Pittman, D. J., and Gordon, C. W.* 255
Types of Drinking Patterns of Male Alcoholics. *Jackson, J. K.* 269
Choice of Alcoholic Beverage Among 178 Alcoholics in Italy. *Lolli, G., et al.* 303
Changing Characteristics of Private-Hospital Alcoholic Patients. A Twenty-Year Time-Trend Analysis. *Maxwell, M. A., et al.* 309
The Rate of Alcoholism in the U.S.A., 1954-1956. *Keller, M., and Efron, V.* 316
Alcoholism and the Reinforcement Theory of Learning. *Kingham, R. J.* 320
The Determination of Ethanol in Biological Materials. *Lester, D., and Greenberg, L. A.* 331

SEPTEMBER

Effect of Insulin on Ethanol Metabolism in Normal and Alloxan-Diabetic Dogs. *Kinard, F. W., and Cox, E. C.* 375
Physiological Alcohol Consumption. *Forsander, O., et al.* 379
The Effect of Ethyl Alcohol on Growth and Voluntary Consumption of Alcohol by Successive Generations of Mice. *Mirone, L.* 388
The Effect of Alcohol on Sexual Reflexes and Sperm Count in the Dog. *Teitelbaum, H. A., and Gantt, W. H.* 394
Anesthesia. LVIII. Biochemical Effects of Acetaldehyde and Other Aldehydes on Oxidative Phosphorylation. *Rehak, M. J., and Truitt, E. B., Jr.* 399
A New Adjunct to the Treatment of Alcoholism: The Hallucinogenic Drugs. *Smith, C. M.* 406
Toxic Effects of Disulfiram Therapy. With Two Case Reports. *Rathod, N. H.* 418
Motivation in the Treatment of Alcoholism. *Lemere, F., et al.* 428
Clinical Evaluation of Blood Pressure in Alcoholics. *Schnall, C., and Wiener, J. S.* 432
Attitudes of Alcoholics Toward Mental Hospitals. *Wolfensberger, W. P.* 447
Religious Affiliation and Drinking Behavior. *Skolnick, J. H.* 452
Alcohol, Amnesia and Awareness. *Washburne, C.* 471
Japanese Drinking Patterns. Alcoholic Beverages in Legend, History and Contemporary Religions. *Yamamuro, B.* 482
Drinking Patterns of the Bolivian Camba. *Heath, D. B.* 491

VOL. 9 QUART. J. EX. PSYCHOL. NOVEMBER, 1957

Periodicity of Response in Operant Extinction. *Hurwitz, H. M. B.* 177
One Trial Learning and the Suppression of Interference. *James, H.* 185
Some Effects of Thiamine Deficiency and Reduced Caloric Intake on Avoidance Training and on Reactions to Conflict. *Khairy, M., et al.* 190
Some Acute Effects of *D*-Lysergic Acid Diethylamide on the Rat. *Glow, P. H.* 206
Transfer Effects in Reactions to "Stress". *Steinberg, H., and Russell, R. W.* 215

VOL. 10 FEBRUARY, 1958

The Measurement of Elements in an Assembly Task—The Information Output of the Human Motor System. *Annett, J., et al.* 1
Some Tests of the Decay Theory of Immediate Memory. *Brown, J.* 12
Blocking the Incorrect Alley in a Two-Drive Learning Situation. *Deutsch, J. A., and Anthony, W. S.* 22
The Effect of Training and Handedness on the Performance of Two Simple Motor Tasks. *Provins, K. A.* 29
Visual Discrimination of Shape by Octopus. Squares and Triangles. *Sutherland, N. S.* 40
Copying Behind During Dictation. *Poulton, E. C.* 48
Studies in Light-Reinforced Behaviour III. The Effect of Continuous Zero and Fixed-Ratio Reinforcement. *Stewart, J., and Hurwitz, H. M. B.* 56

MAY

An Experimental Synthesis of the Associationist and Gestalt Accounts of the Perception of Size. Part 1. *Joynson, R. B.* 65
Visual Illusions Viewed as Stabilized Retinal Images. *Pritchard, R. M.* 77

Some Principles Governing Changes in the Apparent Lightness of Test Surfaces Isolated from their Normal Backgrounds. *Newson, L. J.*	82
Speech Production and the Predictability of Words in Context. *Goldman-Eisler, F.*	96
Guessing, Expectancy and Autonomous Change. *James, H.*	107

AUGUST

The Restriction of Peripheral Vision During Exposure to Hot and Humid Conditions. *Bursill, A. E.*	113
The Effect of Warning Interval on the Electric Phosphene and Auditory Thresholds. *Howarth, C. I., and Treisman, M.*	130
An Experimental Synthesis of the Associationist and Gestalt Accounts of the Perception of Size. Part II. *Joynson, R. B.*	142
A Study of Evaluative Problem Solving. Part I. Observations on Adults. *Rayner, E. H.*	155
The Effect of Small Brightness Differences on the After-effect of Seen Movement. *Day, R. H.*	166

VOL. 44 QUART. J. SPEECH APRIL, 1958

The Speech as Literary Genre. *Murphy, R.*	117
The Debate About Debating. *Ehninger, D.*	128
Richard Sibbes's Theory and Practice of Persuasion. *Hudson, R. F.*	137
The Influence of Style of Moderation on the Outcomes of Discussion. *Utterback, W. E.*	149
Winston Churchill: Orator-Historian. *Lomas, C. W.*	153
John Morley: Critic of Public Address. *Moore, D. E.*	161
Notes Toward a Theory of Diction. *Ostroff, A.*	166
Linguistics and Literary Analysis. *McLaughlin, J. C.*	175
Haiman to Hellman. *Haiman, F. S.*	178
An Exemplary Taft Letter. *Ryan, P. M., Jr.*	178
An Author Objects. *Lowenthal, L.*	179
The Reviewer Explains. *McCollom, W. G.*	180

VOL. 46 RASS. STUDI. PSICHIAT. 1957

Psychopathological and Critical Contribution to the Problem of the Early Diagnosis of Schizophrenia. *Moreno, M.*	812
Mental Disturbance in Pernicious Anemia. *Fabbri, W.*	849
The Value of Hormonal, EEG and Angiocarotidographic Evidence in the Study of the Sturge-Weber Syndrome. *Biasci, L., and Ronco, P.*	859
Cochlear-vestibular Symptoms in Psychoneuroses. *Cannizzaro, C.*	873
A Contribution to the Study of the Metabolism of Indol Compounds, Particularly 5-hydroxyindolacetic Acid in Schizophrenia. *Buscaino, G. A., and Stefanachi, L.*	885
Observations on the Behaviour of Leucotomised Patients. *Mancini, E., et al.*	894
Factor-analysis in Clinical-therapeutic Researches. *Nicoletti, I., and Magherini, G.*	903

VOL. 47 1958

Neurological Syndrome in Malformations of the Cranio-cerebral Bones. *Floris, V., et al.*	1
Possibilities and Limits of Decourt-Doumic's Anthropometric Methods in Clinical Psychiatry. *Pelligrini, G., and Senini, G.*	39
EEG Observations on a Case of Subacute Sclerosing Encephalitis. *Alberton, G.*	49
The Psychodiagnostic Value of Tsedek's Test in Amoral Constitutions During Subnarcosis. *Ronco, P., and Cacopardo, A.*	57
Preliminary Observations on the Action of Meprobamate in Neurological Conditions and in Epilepsy. *Maccagnani, G.*	71
A Contribution to the Treatment of Delirium Tremens. *Inga, E. F., and Sbarbori, F.*	77
Clinical Results with Meprobamate. *Maccaqnani, G.*	96
A Particular Aspect of an Enlarged Cavity in the Septum Pellucidum. *Fattovich, G.*	111
The Therapeutic Problem of Schizophrenia. *Fabbri, W., and Salvatores, U.*	119
A Contribution to our Knowledge of Medico-legal Problems of Minors. *Ragozzino, D.*	189
Research into Intellectual Impairment in Alcoholics. *Bosinelli, M., and Carloni, G.*	209
Problems of Personality in Sanatorium Patients. *Bosinelli, M., and Maccaqnani, G.*	267
An Uncommon and Early Sign of Schizophrenia. *Giove, C.*	297
"Pseudo-senility", an Affective State in the Over-60 Group. *Cibelli, S.*	321
Luescher's Chromatic Test in Childhood. *Pelligrini, G., et al.*	341
Tsedek's Test in Normal Subjects and in Criminal Psychopaths. *Corrado, F.*	357
First Results in the Treatment of Depressions by Iproniazide (Rivivol). *Marzuoli, U.*	401
Mortality in Mental Hospitals in Florence before and after the War. *Nicoletti, I.*	413
The Problems of Symbolism in Mental Disease. *Piazzesi, W.*	413
Otoneurological Consideration in True and False Vertigo. *Canizzaro, C.*	445
The C.S.F. in Senile Dementia. *Albanese, B.*	500

VOL. 4 REV. ALCOOL. 1957

Fourteen Months Survey of Accidents on the Road Eventually due to Alcohol. *Cossa, P., and Lalement, S.*	237

Biochemical Surveys of Acute Alcoholism. *Weil, E.*	256
The Amount of Alcohol in the Blood. *Jaulmes, P., and Mestres, R.*	267
Alcoholemia. *le Polles, C. Y.*	276
Alcoholism and Tuberculosis. *Voisin, R., et al.*	301
Alcoholic Encephalopathies. *Devic, M., and Tomasi, M.*	325
The Use of Antabuse in the Form of Implants in the Treatment of Chronic Alcoholism. *Owsianik, T., et al.*	349
Experimental Researches on the Cutaneous Reactions of Alcoholic Guinea-pigs. *Barriere, H.*	357
Is there a General Fall in the Consumption of Alcohol? *Perrin, P.*	378

VOL. 97 REV. NEUROL. SEPTEMBER, 1957

Extrapyramidal Pathology. *Thiebaut, F.*	163
States of Conscience in Neurology. *Hecaen, H.*	182
Multiple Sclerosis and Demyelinating Diseases. *Lhermitte, F.*	191
The Pathology of Demyelinating Diseases. *Lhermitte, F.*	197
Neuropathology. *Gruner, J.-E.*	202
The Fourth International Congress of EEG and Clinical Neurophysiology. *Fischgold, H., and Blanc, C.*	220
A Histopathological Study of a Case of Invasion of the Nerve Centres and of the Viscera by "Torula histolytica". *Bertrand, I., and Mollaret, H.*	241
The Possibilities of Diagnosis during Life of Cerebral Calcinosis (Fahr's Disease). *Saginario, M., et al.*	251
The Classification of Muscular Stiffness of Cerebral Origin. *Tardieu, G., et al.*	264
The Role of the Cerebellum and of the Brain-stem in the Genesis of Dyskinesias of Extrapyramidal Type. *Aseni, C., and Sterade, M.*	276
Subacute Sclerosing Leucoencephalitis in the Adult. *Wender, M.*	285
Friedreich's Disease with Optic Atrophy, etc. *Nayrac, P., et al.*	295
Sexual Chromatin of Human Neuroglia. *Bertrand, I., and Girard, C.*	321
The Phenomena of Homolateral "Instinctive" Gripping in Posterior Hemispherical Lesions. *Hecaen, H., et al.*	336
A Case of Ferric Deposit in the Central Nervous System. *Garcin, R., and Lapresle, J.*	417
Two Cases of Degeneration of the Corpus Callosum. *Boudin, J., et al.*	433
A Polyradicular Syndrome of Familial Character. *Cossa, P.*	450
Eosinophilia of the Cerebro-spinal Fluid. *Lapon, R., et al.*	466

VOL. 98 1958

Experimental Results Obtained in Human Encephalitis, etc. *Pelc, S., et al.*	3
A Clinical Study of 210 Cases of Intoxication with Organic Salts of Tin. *Alajouanine, Th., et al.*	78
Toxic Encephalopathies with Stalinon. *Cossa, P., et al.*	97
Lesions of the Neuraxis Secondary to the Ingestion of Stalinon. *Gruner, J.-E.*	109
Prolonged Hypersomnia Caused by Bilateral Lesions of the Medial Activator System. *Façon, E., et al.*	117
The Influence of Temporal Lobectomy on the Mental State of Psychomotor Epileptics. *Alajouanine, T., et al.*	165
Little's Disease. *Tournay, A., and Paillard, J.*	172
Encephaloceles, Meningoceles from Hypervitaminosis A and Clinical Considerations. *Giroud, A., et al.*	181
Encephalitis from Asian Flue. *Furtado, D.*	192
A Contribution to the Study of Encephalopathy of Pancreatic Origin. *Bertrand, I., et al.*	245
New Observations of Familial Idiocy with Spongy Degeneration of the Nerve Centres. *de Vries, E., et al.*	271
A Contribution to the Study of Paralysis with Hyperkaliemia. *Mollaret, P., et al.*	341
Kypho-scoliotic Paraplegia. *Rouquès, L.*	358
The Neurological Syndrome of "Delirium Tremens". *Boudin, G., et al.*	381
Central Lesions in von Recklinghausen's Disease. *Jequier, M., and Rabinowicz, T.*	391

VOL. 28 RIV. NEUROL. 1958

The Capillary Resistance in the Arms in Neoplastic Lesions of the Frontal Lobe. *Zappi, F.*	1
Extradural Spinal Cysts and Occlusion of the Foramina of Luschka and Magendie. *Balestrieri, A.*	17
Amaurosis in Epilepsy. *Fiume, S.*	43
Nocturnal Fits in Adults. *Morocutti, C., and Vizioli, R.*	56
Histological Study of the Cerebrum after Convulsive Attacks in Rabbits. *Morocutti, C., and Vizioli, R.*	67
Psychopathological Considerations in Two Cases of Poisoning by Phenylpropylamine Derivatives. *Tolentino, I.*	81
Angiographic Diagnosis of the Nature of Cerebral Tumours. *Floris, V., and Severini, P.*	113

The Appearance of Herpes Simplex During Tetanus and its Favourable Effect on Prognosis. *Montanari, A.* .. 209
The Action of Some Sympathomimetic Substances on Convulsive Attacks from Nicotine in the Rabbit. *Morocutti, C., and Sergio, C.* .. 216
The Effect of Metrazol and Photometrazol on the EEG of 5,000 Epileptics and Other Psychiatric Subjects. *Zamparo, D.* .. 233
Clinico-statistical Considerations on the Visual Changes in Endocranial Meningiomas. *Moscatelli, G. F., and Severini, P.* .. 289
Epilepsy with Aphasic Expression. *Fiume, S.* .. 321
Electrocorticographic Modifications and Variation in Cholesterase in the Midbrain and Hemispheres in Rabbits Treated with Ritalin. *Mariani, E., and Morocutti, C.* 331
Evidence of Disturbance of Metabolism with 5HT in the Blood of Mental Patients. *Poloni, A.* .. 345
Tuberose Sclerosis and Cerebral Tumor. *Novelletto, A., et al.* .. 352
A Case of Pre-senile Dementia Accompanied by Epileptic Crises and with Biopsy Changes of Pick's Disease. *Agostini, L., et al.* .. 376
Alterations in the Central Nervous System of Guinea-pigs Poisoned with Venom. *Agostini, L., and Cantore, G. P.* .. 391
A Pontine Abscess. *D'Orio, R., and Donini, G.* .. 399
A Large-celled Tumour of the Vertebral Column. *Merigliano, D., and Riccio, A.* .. 408
A Contribution to the EEG Study of Angiomatous Malformations of the Brain. *Nicola, G. C., and Torrigiani, G.* .. 436
Neurological Complications in the Cure of Alcoholism with Antabuse. *Fiume, S.* .. 457
The Relation Between Epilepsy and Obsessive Psychoneuroses. *Fiume, S.* .. 465
Voluntary Movement of Affective-ecstatic Type in Lesions of the Left Cerebral Cortex, etc. *Montanari, A.* .. 473
Isolated Paralysis of the Extensor Digitorum Communis Muscle. *Laterza, A., and Sabbadini, G.* .. 485

VOL. 78 RIV. PAT. NERV. MENT. 1957

A Preliminary Contribution to the Histological and Histochemical Study of the Central and Peripheral Nervous System in Acute Poisoning Attributed to Potassium. *Burruano, F.* .. 793
A Final Contribution to the Histological and Histochemical Study of the Central and Peripheral Nervous System in Acute Poisoning Attributed to Potassium. *Burruano, F.* .. 827
Neurophysiopathological Considerations on Catatonia. *Mattioli-Foggia, C., and Cossio, M.* .. 867
Cerebral Pseudo-tumours. *Cordona, F., and Barontini, F.* .. 875
A Case of Congenital Myotonia. *Mattioli-Foggia, C.* .. 889
Aneurysms of the Carotid and its Two Branches. *Manghi, E., et al.* .. 898
Clinical and EEG Experiments with Three Non-barbiturate Hypnotics. *Masciocchi, A.* 920
Experience in Schizophrenia with a New Psychomimetic Drug. *Ferrio, L., et al.* .. 941
Electromyographic Researches on the Deep Reflexes in Acute Anterior Poliomyelitis. *Sala, E., and Valli, M.* .. 957
Morphological Observations on the Acoustic Nerve. *Cannizzaro, C., and Fabiani, F.* 970

VOL. 79 1958

A Contribution to the Modern Theory of the Neurone. *Cazzato, G.* .. 1
Thrombosis of the Internal Carotid from Cranial Injury. *Menchi, D.* .. 27
Critical Considerations of the Pathogenetic Interpretation of Flaccid Paraplegia, etc., in Old People. *Chiaramonti, E.* .. 41
A Case of Congenital Syphilis with Medullary Manifestations of the Amyotrophic Type of Charcot-Marie-Tooth. *Garello, L., and Soriani, S.* .. 54
The So-called Post-traumatic Syndrome. The Significance and Limits of EEG Inquiry. *Borgna, E., and Giannelli, A.* .. 65
Histological and Histochemical Aspects of the Central Nervous System During Experimental Riboflavin Avitaminosis. *Scarlato, G.* .. 84
The Heredity of Enuresis. *Pescetto, G., and Pardera, G.* .. 95
The Choice of Colour as a Method of Psychodiagnostic Test. *Cazzullo, C. L., and Montanini, R.* .. 105
A Case of Cerebello-myoclonic-epileptic Syndrome. *Vico, B., et al.* .. 132
A Reticulo-sarcoma of Primitive Endocranial Development. *Crosato, F., and Zappoli, R.* .. 149
An Anatomo-clinical Study of a Case of Progressive Spinal Muscular Atrophy of Duchenne-Aran Type, etc. *Chiaramonti, E., and Barontini, F.* .. 189
Model Psychoses. *Gastaldi, G., et al.* .. 225
Agenesis of the Corpus Callosum. *de Negri, M., et al.* .. 237
Paget's Bone Disease and Mental Disturbances. *Biasci, L., and Jaria, A.* .. 257
The Histopathology of Venous Alterations during Cerebral Softening. *de Biase, G.* .. 277

Contributions to our Knowledge of Elective Alterations in the Grey Matter. *Chiaramonti, E., and Royer, R.* 405
The Ontogenesis of the Vestibular Nuclei of Man. *Cannizzaro, C., and Fabiani, F.* .. 425
Free Amino Acids in the Normal Cerebrospinal Fluid, etc. *Aiello, G., and Bettinazzi, N.* 480
Psychopathology and Organic Changes During Chlorpromazine Treatment in Psychoses. *Cazzullo, C. L., and Borgna, E.* 498